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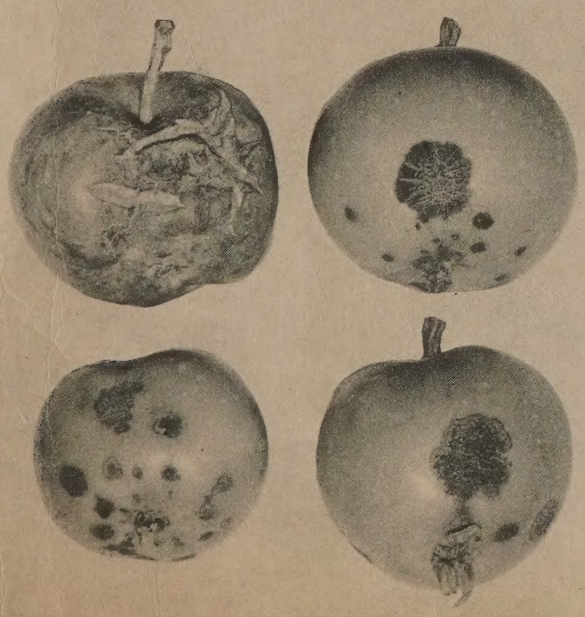
Canadian Plant Disease Survey

Compiled and Edited by D. W. Creelman



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CANADIAN PLANT DISEASE SURVEY

Volume 41 December, 1961 Number 5

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RESISTANCE TO PRE-EMERGENCE DAMPING-OFF IN GARDEN PEAS[1]

W.C. McDonald[2] and H.H. Marshall[2]

Abstract

The resistance of 450 introductions or varieties of peas to pre-emergence damping-off incited by Pythium ultimum Trow. was tested in naturally infested soil and artificially infested sand. In preliminary screening tests 83 per cent of 105 strains with colored flowers and 7 per cent of 345 strains with white flowers were rated resistant. Seventeen colored and 68 white-flowered strains were re-tested in sand culture against a virulent isolate of the pathogen and of these 15 and 7 respectively were significantly more resistant than the check variety Lincoln.

Introduction

Control by chemical seed treatment of pre-emergence damping-off of garden peas, incited by Pythium ultimum Trow., is usually successful and thus little emphasis has been placed on the production of varieties resistant to this disease. Resistant varieties would be valuable to the grower, however, as fungicidal seed treatments are an added expense and inconvenience in the production of this crop. In 1958 a program was undertaken to screen a large number of pea introductions for sources of resistance to P. ultimum to determine whether any might be useful for breeding purposes. This paper presents the results of tests conducted at Brandon, Man. in naturally infested field soil and at Winnipeg, Man. in artificially infested sand.

Materials and Methods

The tests were made with 450 strains of peas which included several local varieties. The samples of seed of introduced strains were obtained from Dr. D.D. Dolan, New York State Experimental Station, Geneva, New York. Samples which appeared to be mixtures of strains were re-selected on the basis of seed characteristics and the entire collection was increased in field plots in 1958. Notes on a large number of plant characteristics such as height, maturity, flower color, seed size and number, etc. were taken.

The initial screening tests for resistance were done in greenhouse beds containing field soil in which poor stands of peas had been obtained.

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[1] Joint contribution from the Research Station, Winnipeg, Manitoba (Contribution No. 98) and the Experimental Farm, Brandon, Manitoba of the Research Branch, Canada Department of Agriculture.

[2] Plant Pathologist, Research Station, Winnipeg, Man. and Head Gardener, Experimental Farm, Brandon, Manitoba.

Each test contained a group of 76 samples. Three replicates, each consisting of 10 seeds of a single strain, were planted. Emerged seedlings were counted 2 weeks after planting. To provide a range of reaction from resistant to susceptible each group contained at least 2 resistant introductions with colored flowers and the variety Lincoln. Lincoln has shown less susceptibility to damping-off than any of the other common varieties of garden peas grown at Brandon and it was used as the check variety in all the tests.

The soil used in the initial tests contained organisms other than P. ultimum so, to prove that the strains selected were resistant to that pathogen, it was necessary to re-test the more promising strains against pure cultures of it. For this purpose the jelly glass technique (1) was employed. Inoculum was prepared from comminuted mycelial cultures grown in 200 ml of 1 per cent pea meal liquid medium for 5 days at 70°F. Five seeds in each glass were inoculated with 40 ml of inoculum diluted 1:74 with distilled water. Four replicates of each strain were planted. Ten days after planting the seedlings were washed free of sand and rated for disease on the following basis: healthy = 100; slight lesioning = 75; tap root firm, side roots rotted = 50; germinated, but root soft = 25; not germinated, decayed = 0. The ratings were totalled for each replicate and divided by the number of seeds planted (excluding hard, not germinated seeds) to obtain a disease index in per cent. Angular transformations were applied to the percentages obtained and an analysis of variance calculated.

Results

In each of the greenhouse tests in naturally infested soil many of the strains appeared to be more resistant than Lincoln. Table 1 shows the occurrence of resistant strains in relation to their geographical origin and flower color. Most of the resistant strains originated in southern Asia or Africa and the majority of them had colored flowers. Many had primitive characteristics such as small, mottled brown seeds. A high proportion of the more susceptible strains with white flowers originated in North America or Australia and undoubtedly trace back to English varieties.

Sixty-four of the most resistant strains with white flowers and 20 of the 87 resistant strains with colored flowers, selected in the preliminary screening, were re-tested with pure cultures of P. ultimum. Of these strains, 22 had significantly greater resistance to P. ultimum than Lincoln (Table 2). The 13 strains with the greatest resistance had colored flowers and brown or purple seed coats. P.I. 180702 was selected previously by Lockwood (2) for partial resistance to Aphanomyces root rot. Of the remainder in Table 2, all but P.I. 196032 (Sel. A) and Vavilov Brown had white flowers and white seed. The high level of resistance in P.I. 180702 as compared to Vavilov Brown, P.I. 167363 (Sel. B) and the Lincoln check is shown in Fig. 1. The tests in artificially infested sand were much more severe than those in natural soil and many of the promising strains selected earlier were as suscpetible as Lincoln in later tests. The resistance shown by the white-flowered strains listed in Table 2 appears to be much greater than that of the variety White Brunswick which Saksena (3) reports to be resistant. That variety did not differ significantly in resistance from Lincoln in these tests.

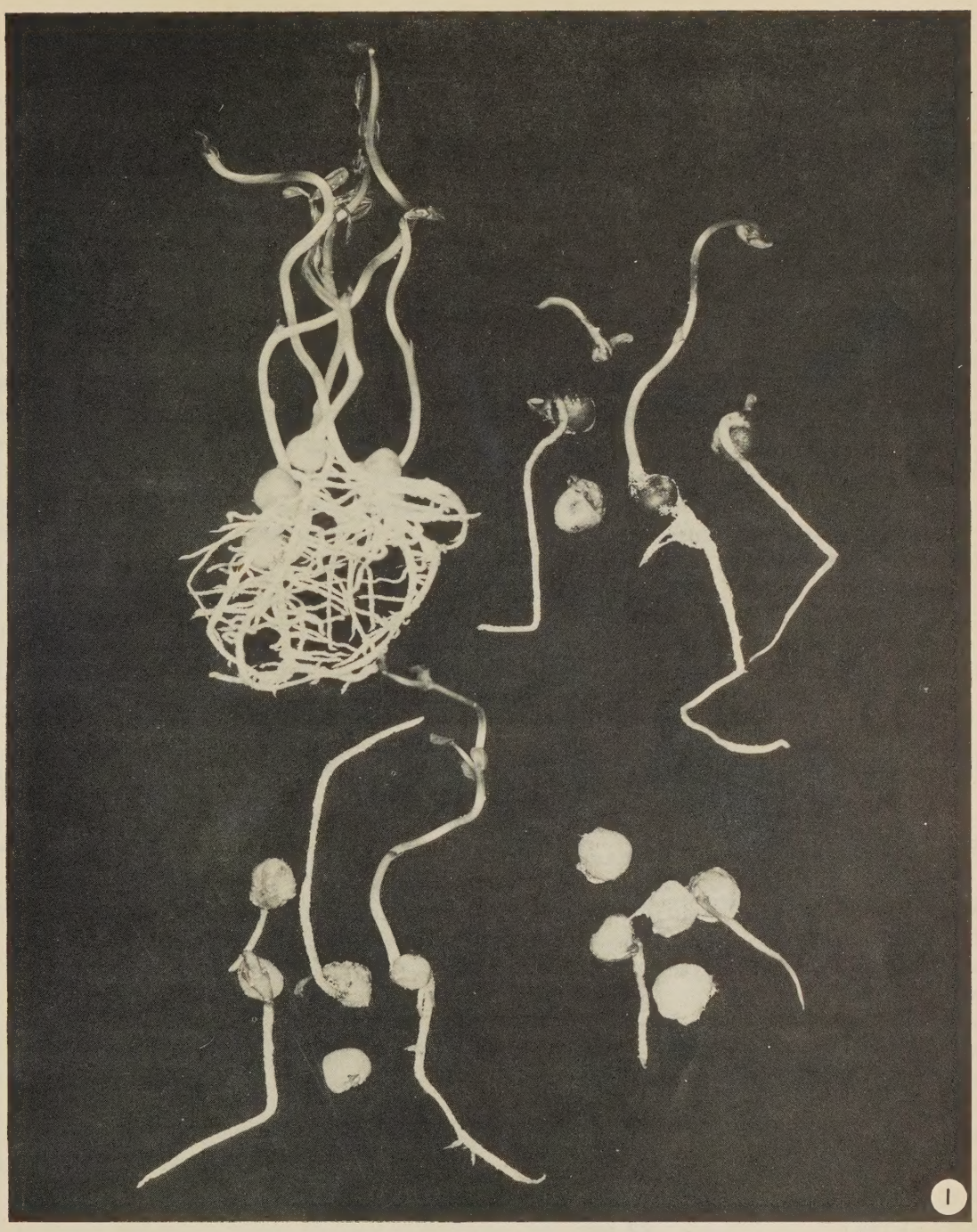

Figure 1. Comparison of pea strains: resistant (upper left, P.I. 180702); moderately resistant (upper right, Vavilov Brown, lower left, P.I. 167363 Sel. B); and susceptible (lower right, Lincoln).

Table 1. Pythium-resistant peas in relation to their place of origin and flower color.

Place of origin[1]	Colored Flowers				White Flowers			
	R[2]	MR	S	Total	R	MR	S	Total
South Asia	23	1	1	25	5	10	19	34
S.W. Asia	28	7	2	37	4	9	29	42
Africa	15	2	1	18	1	5	4	10
N. & E. Asia	0	1	0	1	0	2	7	9
Europe	10	1	0	11	0	4	18	22
S. America	4	0	0	4	2	11	11	24
N. Amer. & Aust.	7	2	0	9	13	46	145	204
Total	87	14	4	105	25	87	233	345
Percent Resistant			83				7	

1/ South Asia - India, Pakistan
South-west Asia - Afghanistan, Iran, Turkey
Africa - Ethiopia
North and East Asia - China, Manchuria, Russia
Europe - Austria, England, Finland, Germany, Netherlands, Sweden
South America - Mexico, Costa Rica, Guatemala, Honduras, Peru, Argentina
North America, Australia - Canada, U.S.A.

2/ R - resistant, MR - moderately resistant, S - susceptible.

Table 2. Disease indices for the most resistant pea strains selected from 86 tested to pure cultures of P. ultimum.

P.I. Number or Variety	Disease Index[1]	P.I. Number or Variety	Disease Index
P.I. 183910	100 **	P.I. 206800 (Sel. C)	79 **
Capuchin type	99 **	P.I. 196032 (Sel. A)	70 *
P.I. 171812 (Sel. B)	97 **	P.I. 123246 (Sel. A)	65 *
P.I. 169606 (Sel. A)	97 **	P.I. 206852	63 *
P.I. 164612 (Sel. A)	96 **	P.I. 195024 (Sel. C)	62 *
P.I. 193843 (Sel. A)	94 **	P.I. 210618	59 *
P.I. 170669 (Sel. A)	90 **	P.I. 206781	58 *
P.I. 234263	90 **	P.I. 210624	57 *
P.I. 180702	87 **	Vavilov Brown	56 *
P.I. 170669 (Sel. B)	85 **	P.I. 167363 (Sel. B)	56 *
P.I. 174922 (Sel. A)	82 **	Lincoln	23
P.I. 210587	82 **		

1/ Mean of 1 to 6 tests

** Significantly better at 1 per cent level than Lincoln in individual tests

* Significantly better at 5 per cent level than Lincoln in individual tests.

Discussion

The predominance of pea strains with colored flowers and grey, brown or purple seed color, in those rated resistant to P. ultimum in the preliminary screening, suggests that resistance may be associated with these characteristics in a manner similar to that found by Sorgel (4) in the relationship between anthocyanin development and resistance to Asochyta diseases of pea. He reported that the resistance in a variety of sugar pea with variegated flowers and red seed to the root rots caused by A. pisi Lib., A. pinodella L.K. Jones, and Mycosphaerella pinodes (Berk. & Blox.) Stone was due to the presence of an anthocyanin in the testa. The strains with colored flowers selected by Lockwood (2) as partially resistant to Aphanomyces root rot also fall into this group. Resistance conditioned by factors contained in colored seed coats would be of little value in producing white-seeded garden varieties. Some of the strains with white flowers and seeds, however, have shown a satisfactory degree of resistance in these tests and may contain factors for resistance different from those conditioning seed color. It is this latter group that should provide adequate sources of resistance to P. ultimum.

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GLOEOSPORIUM ALBUM AND G. MALICORTICIS ON APPLES IN NOVA SCOTIA[1]

C.L. Lockhart[2] and R.G. Ross[2]

Abstract

Gloeosporium album and G. malicorticis cause rots on stored apples in Nova Scotia and have been found in cankers on apple trees. Greenhouse inoculations showed that both species can produce cankers on healthy and injured apple trees. Field inoculations with G. album gave negative results but G. malicorticis readily produced cankers on current and one-year old growth of McIntosh apple trees.

Introduction

Gloeosporium album Osterw. (perfect state Pezicula alba Guthrie) has been recognised as an important cause of apple storage rots in Nova Scotia (4, 8). Recently, Gloeosporium malicorticis Cordley (perfect state (Neofabraea malicorticis Jackson), which has not been previously found in Nova Scotia, has been identified as a cause of a rot of stored apples. Both organisms cause rots or stored apples and cankers on apple trees in other areas (1, 3, 5, 6).

Apart from the fruit-rotting phase little was known about the life history of these organisms in Nova Scotia. Therefore, studies were initiated to determine their occurrence and their ability to cause cankers on apple trees. The results obtained are reported in this paper.

Occurrence

In 1959 a storage rot, caused by G. malicorticis, was found on 1.1 percent of the Cortland apples and on a lesser percent of the McIntosh apples being held in storage to determine the effect of orchard fungicides on the development of storage rots (8). In 1960, Russett and Spy apples from three locations were stored at 38°F. for 5 1/2 months after which they were examined for the occurrence of Gloeosporium rots. The rots caused by Gloeosporium spp. appeared to be of two types. One was larger and of the bull's-eye type. The other was typical of the lenticel type described by Kienholz (7). The two types were kept separate when taking records. The percentage of "bull's-eye rot" present was calculated on whether or not a fruit was infected, regardless of the number of lesions. Identification of the species present was made from spores or from isolations made on an agar medium. The identification of the cause of lenticel rots was made entirely on agar plates.

The data in Table 1 shows that G. malicorticis and G. album were almost equally responsible for the "bull's-eye rot" but the former was more commonly the cause of the lenticel type.

---

[1] Contribution No. 1077 from the Research Station, Canada Agriculture, Kentville, Nova Scotia.

[2] Plant Pathologists.

Table 1. Percentage of G. album and G. malicorticis rots of stored apples

Variety	Location	No. of Apples	Percent bull's-eye type rots caused by G. album	Percent bull's-eye type rots caused by G. malicorticis	Percent lenticel type rots caused by G. album	Percent lenticel type rots caused by G. malicorticis
Russett	Kentville	707	1.5	6.2	2.2	54.5
"	Greenwich	311	1.6	12.5	0	28.1
"	Rockland	591	2.8	7.9	0	65.0
Spy	Greenwich	156	25.6	12.1	20.0	75.0
"	Rockland	325	1.8	0.9	23.0	57.8
Average			6.6	7.9	9.0	56.1

In 1957, G. album was isolated from two cankers occurring at the base of dead one-year old wood on McIntosh trees. Similar cankers, dead spurs, and pruning snags were collected from a number of other orchards in 1958 but attempts to isolate G. album from this material were unsuccessful. In 1960, G. album was isolated from cankers on 10 to 15-year old McIntosh trees located in three different orchards. In two of these orchards the trees were very severely cankered and retarded. These cankers were similar in appearance but more advanced than those shown in Figure 1. G. malicorticis was also present in one of them. It is interesting that both of these orchards had received heavy applications of litter from chicken houses which induces very luxuriant growth late in the season.

Development of cankers in the greenhouse on potted trees

On January 21, 1960, dormant one-year old McIntosh apple trees, which had been removed from storage and potted on January 11, were inoculated by making an incision in the bark with a flamed scapel and inserting a pad of fungus mycelium from an agar culture under the flap. The incisions were wrapped with moistened cotton and held in place with cellulose tape. Controls consisted of incisions without inoculum. Two weeks after inoculation the cotton was removed.

The treatments, each applied to six trees were as follows:

(a) Held in 40°F storage for eight weeks and then transferred to the greenhouse.
(b) Side branches dipped in hot water (161 to 172°F) for two minutes.
(c) Stems girdled below point of inoculation by removing one turn of a 1/16 inch helically cut band of bark.
(d) Watered to excess by daily applications.
(e) Under-watered with only one weekly application.

Two trees from each treatment were inoculated with a culture of G. album (57-1R), two with the same isolate (57-1Ra) which had recently passed through an apple and two with a culture of G. malicorticis. These isolates had originally been obtained from rotted apples. Two inoculations and one control incision were made on each tree.

Observations were made on the cankers as they developed and on April 21 the cankers were removed from the trees and attempts made to recover the fungus in culture. The number of cankers which developed under the various treatments are given in Table 2. No cankers developed in the controls.

The cankers caused by G. album in the greenhouse were slightly sunken, either brown or light-brown in the center with a dark-brown border. Acervuli were sometimes produced on the surface. The cankers caused by G. malicorticis were very similar and varied from light-brown or tan to dark-brown or almost black, usually zonated with wrinkled bark. Acervuli developed more readily than when the organism was G. album.

The data in Table 2 show that G. album and G. malicorticis can be the primary cause of cankers on young McIntosh apple trees. More inoculations were successful with G. malicorticis than with G. album. The number of trees in each treatment was insufficient to detect any differences between treatments but, apparently, apart from the incisions made at inoculation, abnormal conditions are not needed for the development of cankers.

Field inoculation experiments

Inoculation of fruit and leaf spurs:

On September 25, 1959, the fruit and leaves were removed from 15 fruit spurs and the leaves from 15 leaf spurs of each of two young McIntosh apple trees. Ten of each of these two types of spurs on one tree were inoculated with G. album and a similar number of each type of spur on the other tree with G. malicorticis. The wounded tips of the spurs were inoculated by brushing them with a spore suspension of the appropriate fungus prepared from acervuli on rotting apples. Immediately following the application of the spores small polythene bags, containing some water, were placed over the spurs and held in place with elastic bands. The five uninoculated fruit and leaf spurs on each tree were similarly bagged. After two weeks the bags were removed. All inoculated and uninoculated fruit and leaf spurs remained healthy.

Branch inoculations:

In September, 1959, and in June, July, August and October, 1960, current and one-year old wood of young McIntosh apple trees were wound-inoculated using the cotton batting and cellulose tape method described previously. Inoculations were also carried out in May, 1960, but at this time only the previous year's growth was inoculated. Ten limbs of each years' growth were inoculated with G. album and ten with G. malicorticis in each month by placing a piece of rotted apple tissue, from an apple infected with the appropriate organism, in a wound incision. In October, 1960, G. malicorticis from an agar culture was used instead of the rotted apple tissue. Uninoculated wounds were made as before.

No cankers developed in uninoculated wounds or those inoculated with G. album. With G. malicorticis, cankers developed from all inoculations on both current and one-year old wood.

Figure 1. *G. malicorticis* cankers on 1–yr. old wood. Canker on right shows fiddle-string appearance and the one on left shows the start of the fiddle-string appearance.

Table 2. Number of cankers developed on one-year old McIntosh apple trees.

Treatment	Organism		
	G. album	G. album	G. malicorticis
	58-1R	57-1Ra	
Watered daily	1*(1)	2	2 (1)
Watered weekly	1 (1)	1	2 (2)
Eight weeks at 40°F.	2 (1)	0	4 (4)
Hot water	3 (2)	1	3 (1)
Girdled	2 (1)	2 (1)	3

* Out of a total of four inoculations. The number in brackets is the number of cankers from which the organism was isolated.

The average length of the G. malicorticis cankers in mm developing from the 1959 inoculations was as follows:

Date measured	December, 1959	May, 1960
Current year's wood	6.9	8.3
One-year old wood	17.2	20.5

It is obvious that larger cankers developed on the one-year old wood than on the current year's growth and that the cankers increased in size during the winter months. The May examination showed that in a few instances limbs were girdled by the cankers.

The cankers that developed from the 1960 inoculations were measured in May, 1961, and their average length in mm was as follows:

Month inoculated	May	June	July	August	October
Current year's wood	-	10.6	8.6	9.1	13.5
One-year old wood	13.8	16.5	12.4	12.2	15.5

Again the cankers on one-year old wood were larger and by May, 1961, cankers on both types of wood were about the same size regardless of the time of inoculation.

In the early stages of development the cankers in these field experiments were similar to those in the greenhouse but as they developed they became more sunken and usually took on the fiddle-string appearance illustrated in Figure 1. Acervuli of G. malicorticis often appeared on the surface of the cankers one to two months following inoculation but in the advanced stages of canker development acervuli were difficult to find.

Discussion

Apart from their ability to cause apple fruit rots in storage, the role of G. album and G. malicorticis on apple trees in Nova Scotia is not well understood. This study has shown that G. album is present in cankers in the orchard and can cause cankering of healthy and injured apple trees in the greenhouse. Field inoculations with this organism gave negative results. Corke (3) was able to produce cankers by inoculating with G. album in July, August and September but not in other months. Clarkson (2) found G. album to be very prevalent on apple wood following winter injury and considered it to be a saprophyte on dead tissue.

G. malicorticis was also found in cankers in the orchard and cankers were readily produced both in the greenhouse and in the orchard. The importance of this organism in causing cankers of apple trees under natural conditions is not known. Von Arx (1) considered G. malicorticis and G. perennans to be the same species but the perennial cankers caused elsewhere by the latter organism are not present in Nova Scotia.

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INTERNAL SPROUTING OF POTATOES

L.V. Busch[1]

In 1959, a provisional licence, renewable annually, was granted Standard Chemicals, Montreal, Que. for the sale of CIPC or Chloro-IPC (isopropyl N - (3 chlorophenyl) carbamate) for use in potato storages as a sprout inhibitor. The use of this chemical in Ontario in 1960 caused losses of up to 15 per cent due to internal sprouting in stored potatoes. All four chipping varieties examined, Sebago, Kennebec, Irish Cobbler and Idaho Russet were equally affected.

Wherever the disorder appeared symptoms were practically identical, with the sprout, instead of growing out from the tuber eye, turning inward and growing into or completely through the tuber. In many cases the end of the sprout would be swollen to form a small tuber up to one inch in diameter within the old tuber. The mother potato would split in the process and allow the young tuber to partially protrude. (Fig. 1).

The Chloro-IPC was applied on December 5, 1960 at the rate of 1 quart per 1000 bu. as an aerosol calculated to give a concentration in the storage atmosphere of 50 ppm and a concentration on the tuber surface of 1 to 2 ppm. The potatoes were stored at 50°F in pallet boxes 4' x 4' x 4', each box containing approximately 2200 lb. The disorder was first noticed in early March, about mid-way through the storage season and appeared to be more severe in the center of the boxes.

This type of reaction in the potato is not new, having been previously reported by Stewart (1) who found that potatoes which had been left in storage over the summer had produced internal tubers by September. Stuart (2) also noticed that a sample of potatoes which had been placed in a relatively warm, dry room gave rise to similar abnormal sprouts. In addition to these two cases reported in the literature a similiar sample was received by Botany Department, Ontario Agricultural College in June, 1950 from the agricultural representative at Petrolia, Ontario. However, unlike the disorder induced by Chloro-IPC, the previously reported cases of internal sprouting appeared too late in the storage season to cause any appreciable damage and were looked upon as biological curiosities rather than a serious economic problem.

[1] Assistant Professor, Department of Botany, Ontario Agricultural College, Guelph, Ontario.

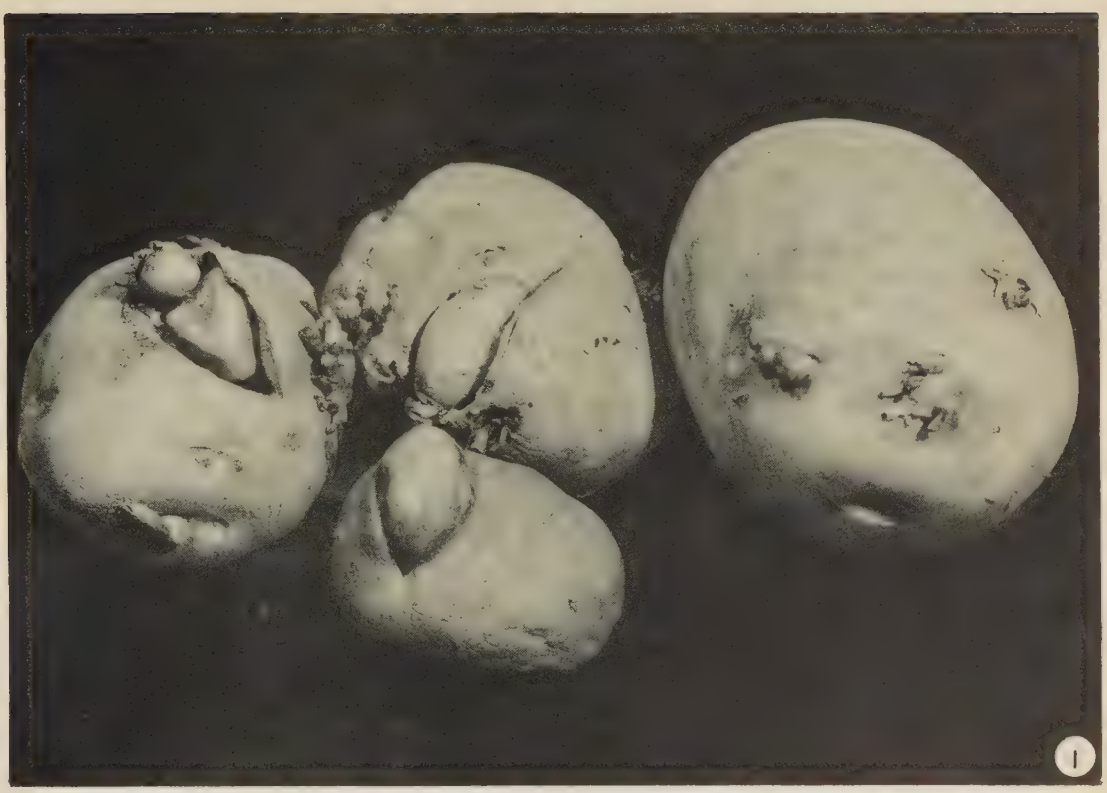

Figure 1. Internal sprouting of potatoes

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DISCOVERY OF THE SUGAR-BEET NEMATODE IN WESTERN CANADA[1]

C.E. Lilly[2], A.M. Harper[2], and E.J. Hawn[3]

This is a report of the first record of the sugar-beet nematode, Heterodera schachtii Schmidt, in Western Canada. It had previously been found in eastern Canada in two localized areas of southern Ontario, near St. Catharines in 1931 and near Sarnia in 1939.

On June 22, 1961, members of the research staff of the Canadian Sugar Factories drew the attention of the two senior authors to unthrifty sugar beets in an irrigated field near Taber, Alberta. The field was approximately 13 acres in size and the beets on 4 acres were severely stunted and the leaves badly wilted. Numerous white cysts were found on the roots of the stunted plants as well as on the roots of the other sugar beets throughout the field. There was a greatly increased development of small lateral rootlets on the stunted beets, giving a whiskered appearance to the roots. Cysts were also found on mustard and on oak-leaved goosefoot, Chenopodium glaucum L., in the same field. The cysts were identified as those of H. schachtii by Dr. A.D. Baker and R.H. Mulvey, Nematology Section, Entomology Research Institute, Canada Department of Agriculture, Ottawa, Ontario.

In early July sugar beets and soil from the most unthrifty portions of 721 fields throughout southern Alberta were examined in the laboratory for cysts of the nematode but no other infestations were found.

The infested field was ploughed, fumigated with 25 gallons per acre of Shell DD, and planted to alfalfa. Crops that are hosts for H. schachtii will not be grown on this land for 6 years. On the rest of the infested farm a 4-year rotation will be started in which sugar beets or any other susceptible crop will be grown only once every 4 years. Officials of the Sugar Beet Growers' Association and the Canadian Sugar Factories Limited have agreed to adhere to a 4-year rotation in all sugar-beet growing areas of Alberta to prevent serious infestations of this pest developing.

CANADA AGRICULTURE RESEARCH STATION,
LETHBRIDGE, ALBERTA.

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[1] Contribution from Canada Agriculture Research Station, Lethbridge, Alberta.

[2] Entomologist.

[3] Plant Pathologist.

AN OCCURRENCE OF HETERODERA TRIFOLII GOFFART, 1932 IN THE NIAGARA PENINSULA, ONTARIO[1]

J.L. Townshend and R.H. Mulvey[2]

Larvae of a species of Heterodera were noted among nematodes extracted by the Oostenbrink "cottonwool filter" method (3) from the soil of vegetable plots at the Ontario Horticultural Experiment Station, Vineland Station, in the Niagara Peninsula. Further samples were then collected from the same plots and from an adjacent vegetable area sown to a cover crop of Ladino clover. These samples were processed by the Fenwick method (1) and 35 and 40 cysts per 200 grams of soil from the two areas respectively were extracted. Most of the cysts from both areas were old and badly deteriorated. The nematode was identified as Heterodera trifolii Goffart, 1932. This report constitutes the first record of that nematode in the Niagara Peninsula.

Ladino clover, a host of H. trifolii (2), is used in a four-year rotation in the Ontario Horticultural Experiment Station vegetable plots in the Niagara Peninsula. When a portion of the vegetable growing area is in cover crop, Ladino clover is sown together with either oats or rye. The crop is mowed until the clover predominates during the first season and the clover is ploughed down at the end of the second season. Thus the cereal crop occurs one year in four and clover two years in four. Heterodera trifolii apparently is able to survive in this rotation. However, the nematode does not appear to have a detrimental effect on the clover.

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[1]Contribution No. 22, Research Laboratory, Research Branch, Canada Department of Agriculture, Vineland Station, Ontario.

[2]Entomology Research Institute, Research Branch, Canada Department of Agriculture, Ottawa.

EXPERIMENTS ON THE CHEMICAL CONTROL OF SNOW MOLD IN TURF

S. G. Fushtey[1]

Abstract

Seven commercial fungicidal preparations were tested for their effectiveness in the control of snow mold on a naturally-infected golf green. Single applications of all the materials tested proved to be effective when used at the following rates per 1000 square feet: Actidione-Thiram Turf Fungicide, 6 ounces; Calo-Chlor, 3 ounces; Merfusan, 2 to 3 pounds; Mersil, 4 to 6 ounces; Panogen Turf Spray, 3 to 4 ounces; Puraturf, 3 ounces; and Tersan OM, 6 ounces.

Introduction

Snow mold is a troublesome disease of grasses in lawns and in golf and bowling greens in most parts of Canada. In response to numerous inquiries regarding the control of snow mold, the author carried out small-scale experiments in 1959-60 and 1960-61 in order to determine the effect and value of some of the recommended commercial turf fungicides under conditions prevailing at Guelph, Ontario.

The disease causes unsightly dead patches of grass early in the season as illustrated in Figures 1, 2 and 3. The turf usually recovers in May or June but in the meantime it is unsightly and rough. The affected areas, as observed at Guelph, are of two distinctly different types:

(1) Small, circular spots with sharp, brownish margins. These overlap to form irregular areas of pale colored, dead leaves. (Figure 2).

(2) Larger, more regularly circular spots with the pale, dead-appearing areas mostly confined to a band or halo surrounding an area of less severely affected grass which appears quite green. (Figure 3). These spots are several times larger than those described in (1); the damage does not appear as severe, and recovery is more rapid.

Studies are underway to determine the exact cause or causes of the different symptoms. The present paper is confined to the study of the effect of fungicidal treatments in the control of the snow mold complex as it occurs in the Guelph area.

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Experimental Results

The experiments were conducted on the fourteenth green of Cutter Golf Club at Guelph; a green that has been affected annually by snow mold. No artificial inoculum was added.

The fungicides tested, their manufacturers, and the active ingredients were:

1. Actidione - Thiram Turf Fungicide --- Cycloheximide, 0.75%; thiram 75%
   The Upjohn Co., Kalamazoo, Mich.
2. Calo-Chlor --- Mercuric chloride, 28%; mercurous chloride, 57%.
   Mallinchrodt Chemical Works, Ltd., Montreal, Que.
3. Merfusan --- Mercuric chloride, 2.15%; mercurous chloride, 5%; ferrous sulphate, 0.70%.
   May and Baker, Ltd., Dagenham, England.
4. Mersil --- Mercuric chloride, 15%; mercurous chloride, 30%.
   May and Baker, Ltd., Dagenham, England.
5. Puraturf --- Phenyl mercury acetate, 10%.
   Gallowhur Chemicals Canada, Ltd., Montreal, Que.
6. Panogen Turf Spray --- Methyl mercury dicyandiamide, 2.2%.
   Morton Chemical of Canada, Ltd., Winnipeg, Man.
7. Tersan OM --- Thiram, 45%; hydroxymercurichlorophenol, 10%
   E.I. Dupont de Nemours, Wilmington, Del.

In 1959-60 only 3 preparations were tested. The experiment comprised 32 plots, each 5x10 feet in area, randomized in 4 replications. The fungicides were mixed with Milorganite (activated sewage sludge with 5 per cent nitrogen) and the mixture applied with a fertilizer spreader at the rate of 3 lb/1000 sq. ft. The plots were treated on November 26, 1959 and disease index ratings were recorded on April 22, 1960. The results are shown in Table 1.

Table 1. Effect of fungicides on snow mold of turf, 1959-60.

Fungicide	Dosage /1000 sq. ft.	*Disease Index
Mersil	3 oz	** 2.5
	4 oz	1.75
Puraturf	1 oz	3.0
	2 oz	2.25
Tersan OM	6 oz	1.0
	8 oz	1.0
Check (Milorganite alone)	---	3.25

* Disease Index - average of 4 replications.

** Rating - 1 - less than 5 per cent of area affected (trace)
2 - 5 to 20 " " " " "
3 - 20 to 35 " " " " "
4 - 35 to 50 " " " " " (severe)

Figure 1. Snow mold damage on an untreated golf green.
Apron to left and green to right of center.

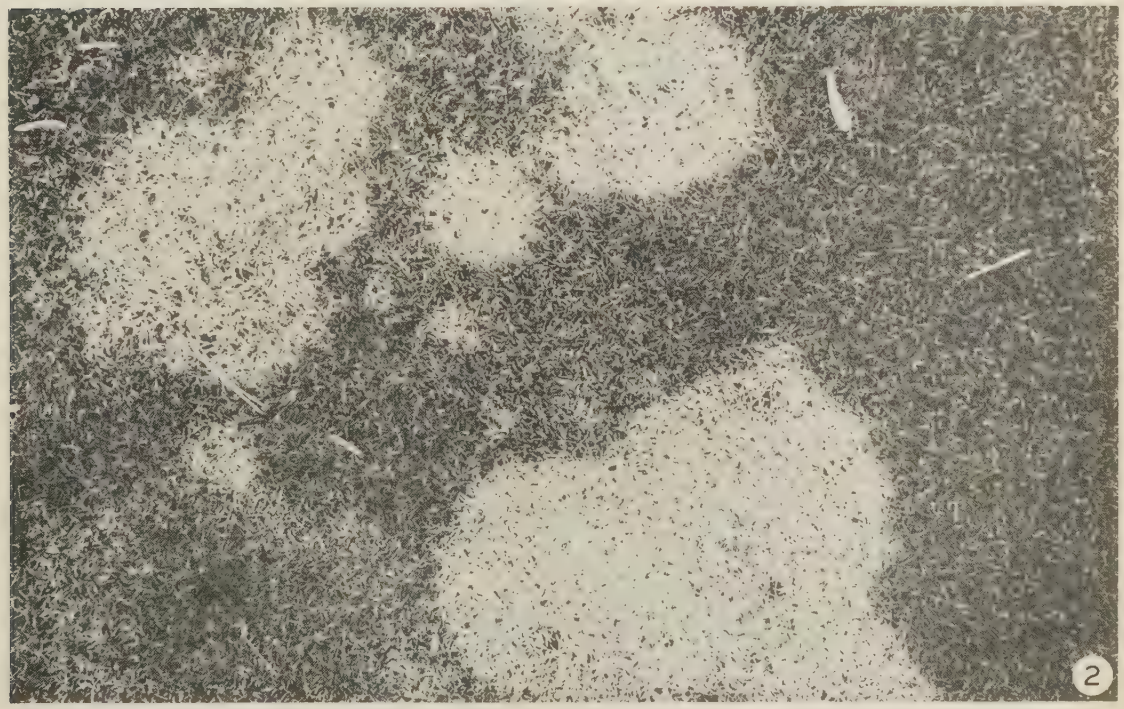

Figure 2. Spots with sharp, brownish margins.

Snow mold was severe in 1960 with approximately 30 per cent of the plot area affected in the untreated check plots. Tersan OM was the only material that give complete control in any of the plots and sufficient overall control to be regarded as effective for practical use. The striking difference between the appearance of an untreated plot and one treated with Tersan OM is illustrated in Figure 4. Mersil and Puraturf gave some control at the higher dosage rates but the level of effectiveness was inadequate for practical consideration.

In 1960-61 seven fungicides, including the 3 tested the previous season, were tested in the same site. In this instance they were applied as a spray in water solution or suspension on December 7, 1960 and ratings were recorded on April 20, 1961. The results are recorded in Table 2. A revised index for rating the amount of disease present was used in 1961, hence the results presented in Tables 1 and 2 are not directly comparable. They must be interpreted in terms of the estimated damage as given in the rating legend for each table.

Table 2. Effect of fungicides on snow mold of turf, 1960-61.

Fungicide	Dosage /1000 sq. ft.	*Disease Index
Actidione-Thiram	6 oz	** 1.0
Turf Fungicide	8 oz	*** 1.5
Calo-Chlor	3 oz	0.75
	4 oz	0.75
Merfusan	2 lb	1.5
	3 lb	0.5
Mersil	4 oz	1.0
	6 oz	0.5
Puraturf	3 oz	1.25
	4 oz	1.25
Panogen Turf Spray	3 oz	1.25
	4 oz	1.0
Tersan OM	6 oz	0.75
	8 oz	1.0
Check (no fungicide)	---	3.0

* Disease Index - Average of 4 replications

** Rating - 0 - no damage
1 - 1 or 2 small spots (trace)
2 - more than 2 spots but less than 5% affected
3 - 5-20% affected
4 - 20-50% affected

*** Disease index of less than 1.5 was considered effective control.

Snow mold was considerably less in 1961 than in 1960; no more than 10 per cent disease was observed in the untreated plots as compared to 30 per cent the previous year. All the fungicides applied at the dosage rates shown in Table 2 gave significant disease control. A comparison of the

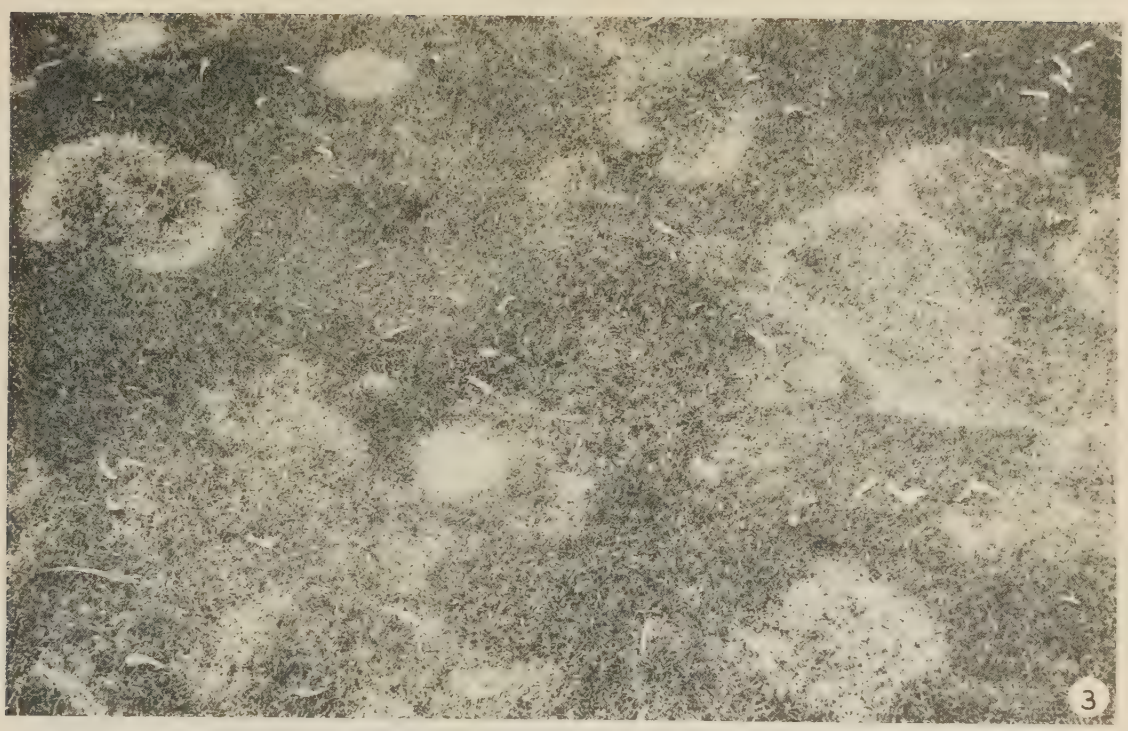

Figure 3. Halo-like spots.

Figure 4. Effective snow mold control with Tersan OM. 6 oz./1000 sq. ft. Treated plot in right foreground. Untreated plot to left of stakes.

results obtained in 1961 with those of the previous year suggests that the poor performance of Mersil and Puraturf in 1960 was due to inadequate dosage rates. It is recommended that the dosage of Mersil be increased to 4 or 5 ounces, and Puraturf to at least 3 ounces, per 1000 sq. ft.

Discussion

The results of the two tests showed that reasonably good control of snow mold can be obtained with a single application of fungicide in the late fall, provided that an adequate amount of fungicide is used. In most cases the dosage rates recommended by the manufacturer were effective. Puraturf, however, did not give adequate control at the recommended 2-oz rate but gave reasonably good control at the 3-oz rate in 1961. On the other hand, Tersan OM was equally as effective at the 6-oz rate as it was at the recommended 8-oz level, thus the lower dosage appears to be adequate.

The use of the Actidione-Thiram formulation for snow mold control requires further investigation because, although disease control was reasonably good, there was evidence of phytotoxicity. The injury did not appear to be serious but plots treated with this material, especially at the 8-oz rate, showed a general tendency to develop a brownish discoloration of the foliage.

Most suppliers of fungicides for snow mold control recommend a second application in mid-or late winter at approximately one-half the initial dosage rate. This recommendation applies to areas where winter thaws and rains remove the snow cover and tend to leach away the fungicide. Most of the damage from snow mold in the Guelph area occurs in late winter and early spring so that additional protection at that time of year would appear to be highly desirable although no supporting data for this recommendation are available at the present time.

Acknowledgements

The cooperation of the management and greenskeeper of Cutter Golf Club in making one of their greens available for use as an experimental area is hereby acknowledged.

DEPARTMENT OF BOTANY
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THE CROWN GALL ORGANISM IN NOVA SCOTIA

C.O. Gourley[1] and K.A. Harrison[1]

Crown gall, caused by Agrobacterium tumefaciens (E.F. Sm. & Towns.) Conn, has been found on a number of hosts in Nova Scotia. The Kentville herbarium contains specimens collected in this province on apple (KP-925), Lombadry poplar (KP-923), willow (KP-1456), rose (KP-1260), rhubarb (KP-263), dahlia (KP-341), English walnut (KP-1640), rutabaga (KP-1696), marigold (KP-991), blackberry (KP-77), raspberry (KP-1788) and rhododendron (a seedling of a cross between Rhododendron smirnowii Trautv. x R. catawbiense Michx. var. Dr. Dresselhuys), (KP-2403). The latter specimen was collected in August, 1961 and is believed to be the first report of crown gall on this host in North America. It has also been reported from Nova Scotia on highbush blueberry[2].

In this district crown gall, although present in small amounts on a number of susceptible hosts, has not been considered to be of economic importance. Certified red raspberry canes from a 5-year-old plantation in light, sandy loam soil, sold in the spring of 1961 to propagating nurseries, developed crown gall on 100 per cent of the canes set. In the propagating nurseries several canes showing unthrifty growth had well developed galls and incipient galls were found on all canes examined. A subsequent examination of the original 5-year-old plantation showed that all plants were infected with crown gall. The varieties Carnival, Early Red, Newburg, Trent, Viking and Willamette were equally affected. This disease has eliminated the sources of certified raspberry plants in Nova Scotia until new disease-free plantings can be established.

In the spring of 1961, after digging the certified canes from the 5-year-old plantation, the remaining canes were mowed off, removed, and destroyed. Irrigation from a surface pond in the near vicinity of the planation kept the new canes in a vigorously growing condition. Suckering was normal and outward appearances did not indicate that gall infections were present. The crowns of several of the tallest, normal looking canes which were leaning over were found to be severely galled and disintegrating. The leaning canes were almost rotted off at the base. Incipient galls were found on the roots of all but a few of the sucker plants.

Other instances of severe crown gall infections of red raspberry have been noticed during the past two growing seasons. Several seedling selections set in old orchard land became severely galled. In the spring of 1961 one seedling selection in a propagation row developed galls on every bud just as they were breaking.

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[1] Plant Pathologists, Canada Agriculture Research Station, Kentville, Nova Scotia.

[2] Lockhart, C.L. in 37th Ann. Rept. Can. Plant Dis. Survey p. 107. 1958.

The reason for the appearance of extensive crown gall infections is not clear. Propagation by digging and transplanting raspberry sucker canes provides numerous points of entry for the crown gall organism. Low rainfall and high temperatures for the past two growing seasons may have provided more favorable environmental conditions for the development of crown gall.

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THE OCCURRENCE OF PYRENOPHORA TERES ON BARLEY STRAW IN ALBERTA

L.J. Piening [1]

In July, 1961, mature ascocarps of Pyrenophora teres (Died.) Drechs., the perfect stage of Drechslera teres (Sacc.) Shoemaker (Helminthosporium teres Sacc.), were found on straw of Olli barley which had been lying in the field since the harvest of 1960. Subsequently they have been found on barley straw from many fields in the Calgary and Edmonton regions indicating their general occurrence. Crowell (1) reported the finding of perithecia of P. teres in the field in Quebec in 1940.

Figure 1. Mature ascocarps on over wintered barley straw.

Figure 2. Mature eight-spored ascus of *Pyrenophora teres*

The setose ascocarps, which are 1-2 mm in diameter, are found on the culm, sheath and nodes of exposed barley straw (Fig. 1). Asci in the fertile fruiting structures normally contain eight multicelled ascospores, though fewer than eight are evident in some asci. The hyaline asci are approxi-

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mately 200-240 μ long and 30-40 μ wide with tough, elastic walls that are difficult to rupture. The multicellular ascospores are light brown in color and measure 44-60 x 16-24μ. (Fig. 2) After four hours on a suitable medium, one or more cells of the ascospore germinate. Conidia typical of D. teres are produced in five days on the mycelium from the germinating ascospores. These conidia incite net blotch lesions on susceptible barley and the conidia produced in these lesions are in turn typical of D. teres.

On V-8 agar some of the monoascosporic cultures differed from others in the number of conidia produced, color of the culture, and in the numbers of sterile ascocarps produced.

Mature ascocarps were also found in 1961 on barley straw grown in 1959. The ascocarps successfully resist weathering and it is possible that spores produced in such ascocarps can cause primary infection of barley grown on a field several years after an infected barley crop was produced.

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CO-OPERATIVE SEED TREATMENT TRIALS -- 1961[1]

J.E. Machacek and H.A.H. Wallace[2]

Abstract

In 1961, twenty-eight seed-treatment products were tested at a number of stations against common bunt of wheat (mixed Tilletia toetida (Wallr.) Liro and T. caries (DC.) Tul.), oat smut (mixed Ustilago avenae (Pers.) Rostr. and U. kolleri Wille), covered smut of barley (U. hordei (Pers.) Lagerh.) and seed rot of flax. All but four were effective against bunt, all but eight were effective against oat smut, and all but six against barley smut. Seven of the treatments significantly reduced seed rot of flax.

Materials and Methods

Kind of Treatment Tested

1. Control -- Seed not treated.
2. Ceresan M -- A powder containing 3.2% mercury as ethyl mercury p-toluene sulfonanilide. Obtained from E.I. duPont de Nemours, Wilmington, Delaware.
3. Leytosan Dual Seed Dressing -- A powder containing 40.0% heptachlor and 2.0% mercury as mixed phenyl mercury acetate and ethyl mercury chloride. Obtained from Leytosan (Canada) Limited, Winnipeg, Manitoba.
4. ACS Mercury Seed Treatment -- A powder containing 5.0% mercury as mixed phenyl mercury acetate and ethyl mercury chloride. Obtained from Allied Chemical Services, Calgary, Alberta.
5. Thiourea Omadine -- A powder containing 50.0% of a derivative of pyridinethione. Obtained from Olin Mathieson Chemical Corporation, Port Jefferson Station, New York.
6. Zinc Omadine -- A powder containing 50.0% of a zinc salt of pyridinethione. Obtained from Olin Mathieson Chemical Corporation, Port Jefferson Station, New York.
7. Ortho LM Dry -- A powder containing 3.2% mercury as methyl mercury 8-hydroxyquinolinate. Obtained from Ortho Agricultural Chemicals Ltd., Vancouver, B.C.
8. Fairview Mercury Compound -- A powder containing 5.0% mercury as mixed phenyl mercury acetate and ethyl mercury chloride. Obtained from Fairview Chemical Company Ltd., Regina, Sask.

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[1] Contribution No. 102 from the Canada Department of Agriculture Research Station, Winnipeg, Manitoba.

[2] Principal Plant Pathologist and Associate Plant Pathologist, respectively, Plant Pathology Laboratory.

9. Seventy-Seven Mercury Seed Dressing -- A powder containing 5.0% mercury as mixed phenyl mercury acetate and ethyl mercury chloride. Obtained from Seventy-Seven Oil Company Ltd., Lethbridge, Alberta.
10. Aagrunol LS 200 -- A liquid containing 2.0% mercury as methyl mercury benzoate. Obtained from N.V. Aagrunol Chemical Works, Groningen, Holland.
11. Aagrunol LSV 150 -- A liquid containing 1.5% mercury as methyl mercury benzoate. Obtained from N.V. Aagrunol Chemical Works, Groningen, Holland.
12. Aagrunol LS 175 -- A liquid containing 1.75% mercury as methyl mercury benzoate. Obtained from N.V. Aagrunol Chemical Works, Groningen, Holland.
13. Aagrunol LSA 175 -- A liquid containing 1.75% mercury as methyl mercury benzoate. Formula different from that of Treatment 12. Obtained from N.V. Aagrunol Chemical Works, Groningen, Holland.
14. Canuck Mercury-Heptachlor -- A liquid containing 25.0% heptachlor and 1.08% mercury as phenyl mercury acetate. Obtained from Gallowhur Chemicals Canada Ltd., Lachine, Quebec.
15. Standard Formaldehyde -- A liquid containing 37.0% formaldehyde. Obtained from Standard Chemical Ltd., Montreal, Quebec.
16. Panogen 15 -- A liquid containing 1.5% mercury as methyl mercury dicyandiamide. Obtained from Morton Chemical Company, Woodstock, Illinois.
17. Pandrinox -- A liquid containing 24.4% heptachlor and 0.5% mercury as methyl mercury dicyandiamide. Obtained from Morton Chemical Company, Woodstock, Illinois.
18. EP-193 -- A liquid containing a mixture of pentachlornitrobenzene and heptachlor. Obtained from Morton Chemical Company, Woodstock, Illinois.
19. Liquid Mergamma -- A liquid containing 2 5/8 lb. heptachlor per Imp. gal. and 0.5% mercury as methyl mercury dicyandiamide. Obtained from Chipman Chemicals Ltd., Hamilton, Ontario.
20. Agrosol -- A liquid containing 1.5% mercury as methyl mercury nitrile. Obtained from Chipman Chemicals Ltd., Hamilton, Ontario.
21. Metasol MP -- A liquid containing 1.25% mercury as methyl mercury propionate. Obtained from Metalsalts Corporation, Hawthorne, New Jersey.
22. ACS Liquid Mercury ST (concentrate) -- A liquid containing 4.2% mercury as methyl mercury 8-hydroxyquinolinate. Obtained from Allied Chemical Services Limited, Calgary, Alberta.
23. ACS Liquid Mercury ST -- A liquid containing 1.25% mercury as methyl mercury 8-hydroxyquinolinate. Obtained from Allied Chemical Services Ltd., Calgary, Alberta.
24. Fairview Liquid Mercury SD -- A liquid containing 1.25% mercury as methyl mercury 8-hydroxyquinolinate. Obtained from Fairview Chemical Company Limited, Regina, Saskatchewan.
25. Niadual -- A liquid containing 25.0% aldrin and 1.45% mercury as phenyl mercury acetate. Obtained from Niagara Brand Chemicals Ltd., Burlington, Ontario.
26. New Gallotox -- A liquid containing 4.1% mercury as phenyl mercury acetate. Obtained from Niagara Brand Chemicals Ltd., Burlington, Ontario.

27. Canuck Liquid Mercury -- A liquid containing 3.88% mercury as phenyl mercury acetate. Obtained from Gallowhur Chemicals Canada Ltd., Lachine, Quebec.
28. Memmi -- A liquid containing 10.0% methyl mercury chlorendimide. Obtained from Velsicol Chemical Corporation, Chicago, Illinois.
29. Morven -- A liquid containing 27.4%, 2,4,5-trichlorophenate. Obtained from Dow Chemical Company, Midland, Michigan.

Kinds of Seeds Used in Trials

Wheat bunt trials -- Variety Red Bobs. Seed artifically contaminated (1:200, by weight) with mixed spores of Tilletia tritici and T. foetida.

Oat smut trials -- Variety Vanguard. Seed naturally contaminated with mixed spores of loose and covered smut.

Barley smut trials -- Variety Plush. Seed naturally contaminated with spores of covered smut.

Flax seed-rot trials -- Variety Redwood. About 50% of the seeds cracked during threshing.

Kinds of Plots and Analysis of Experimental Data

The field plots used in the comparison of treatments were single-row units with the rows 10 to 14 feet long and 9 to 12 inches apart. The units were arranged in randomized blocks with four replicates for each treatment. In supplementary tests under greenhouse conditions the plots were single-row units with the rows about 30 inches long and about 2 inches apart. The field plot experiments were used for the determination of the amount of surface-borne smut in wheat, oats and barley, and for the measurement of non-emergence in flax. The greenhouse tests were used principally for the determination of phytotoxicity of the various treatments to treated seed which was stored in closed glass jars for about 4 1/2 months.

Experimental Results

The data obtained from field plots during 1961 are summarized in Table 1. They show that Thiourea Omadine, Pandrinox and Memmi were moderately effective against wheat bunt while Morven was ineffective. All the other products tested gave good control. With oat smut, Memmi and Morven gave poor control; Thiourea Omadine, Canuck Mercury-Heptachlor, EP-193, Niadual, New Gallotox and Canuck Liquid Mercury gave moderate control, while the remainder of the products tested gave good control. Thiourea Omadine, EP-193 and Morven gave poor control of barley smut, while Niadual, New Gallotox and Memmi showed moderate control. All the other products were satisfactory. In the case of flax, seven of the products tested controlled seed rot fairly well but in most cases there was only a slight control.

In seed that was treated and sown after 4 1/2 months in closed storage some phytotoxicity to wheat was shown by Canuck Mercury-Heptachlor, and severe phytotoxicity by Formaldehyde. None of the treatments seemed to be phytotoxic to oats, barley or flax.

Acknowledgments

The writers wish to thank the following for their excellent help during 1961: Dr. D.C. Arny, University of Wisconsin, Madison, Wisc.; Dr. T.G. Atkinson, C.D.A. Research Station, Lethbridge, Alta.; Mr. H.R. Ballantyne, C.D.A. Experimental Farm, Melfort, Sask.; Mr. J.A. Browning, Iowa State College, Ames, Iowa; Dr. W.P. Campbell, C.D.A. Plant Pathology Laboratory, Edmonton, Alta.; Mr. J.E. Campbell, C.D.A. Experimental Farm, Charlottetown, P.E.I.; Mr. T.C. Chiasson, C.D.A. Research Sation, Fredericton, N.B.; Dr. W. Crosier, Agr. Exp. Station, Geneva, N.Y.; Dr. S.G. Fushtey, Ont. Agr. College, Guelph, Ont.; Dr. A.A. Guitard, C.D.A. Experimental Farm, Beaverlodge, Alta.; Dr. A.W. Henry, University of Alberta, Edmonton, Alta.; Dr. M. L. Kaufmann, C.D.A. Experimental Farm, Lacombe, Alta.; Dr. R.O. Lachance, C.D.A.Research Station, Ste. Anne de la Pocatiere, Quebec; Mr. K.B. Last, Genetics and Plant Breeding Research Institute, Ottawa, Canada; Mr. D.S. McBean, C.D.A. Experimental Farm, Swift Current, Sask.; Mr. M.B. Moore, Institute of Agriculture, University of Minnesota, St. Paul, Minn.; Dr. L.H. Purdy, Regional Smut Research Lab., State College, Pullman, Washington; Dr. R.C. Russell, C.D.A. Research Station, Saskatoon, Sask.; Prof. T.C. Vanterpool, University of Saskatchewan, Saskatoon, Sask.

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Table 1. Co-operative Seed Treatment Trials - 1961. Summary of results[a]

Treatment No.	Abbreviated name of seed dressing	Dose (oz./bu.)[b] Wheat	Oats	Barley	Flax	Percentage smut Wheat	Oats	Barley	Flax	Percentage non-emergence
1	Control (dry, untreated seed)	0.00	0.00	0.00	0.00	33.7	11.8	7.4	63.0	
2	Ceresan M (powder)	0.50	0.50	0.50	1.50	0.1	0.1	0.2	56.2	
3	*Leytosan Dual Seed Dressing (powder)	2.00	1.40	1.40	5.00	0.3	0.3	0.5	58.0	
4	ACS Mercury Seed Treatment (powder)	0.50	0.50	0.50	1.50	0.2	0.2	0.2	55.3	
5	Thiourea Omadine (powder)	0.50	1.00	1.00	3.00	1.2	3.1	6.5	58.7	
6	Zinc Omadine (powder)	1.50	2.00	2.00	6.00	0.1	0.5	0.1	57.3	
7	Ortho LM Dry (powder)	0.50	0.50	0.50	1.50	0.0	0.1	0.2	50.8	
8	Fairview Mercury Compound (powder)	0.50	0.50	0.50	1.50	0.1	0.4	0.3	54.5	
9	Seventy-seven Mercury Seed Dressing (powder)	0.50	0.50	0.50	1.50	0.2	0.0	0.5	54.6	
10	Aagrunol LS 200 (liquid)	0.75	0.75	0.75	1.50	0.0	0.1	0.1	46.2	
11	Aagrunol LSV 150 (liquid)	0.75	0.75	0.75	1.50	0.1	0.1	0.2	49.6	
12	Aagrunol LS 175 (liquid)	0.75	0.75	0.75	1.50	0.0	0.1	0.1	49.5	
13	Aagrunol LSA 175 (liquid)	0.75	0.75	0.75	1.50	0.0	0.1	0.3	53.3	
14	*Canuck Mercury-Heptachlor (liquid)	3.00	2.25	3.00	5.00	0.4	1.4	0.5	62.6	
15	Formaldehyde (liquid)	(Ten-minute soak in 1/320 solution)			(Flax not treated)	0.1	0.2	0.7	63.6	
16	Panogen 15 (liquid)	0.75	0.75	0.75	1.50	0.0	0.0	0.1	53.2	
17	Pandrinox (liquid)	2.12	2.12	2.12	4.00	1.1	0.1	0.1	57.7	
18	EP-193 (liquid)	2.25	2.25	2.25	6.00	0.4	3.9	7.8	63.2	
19	*Liquid Mergamma (liquid)	3.00	1.88	1.88	7.50	0.1	0.1	0.1	57.8	
20	Agrosol (liquid)	0.75	0.75	0.75	1.50	0.1	0.1	0.1	48.9	
21	Metasol MP (liquid)	0.75	0.75	0.75	1.50	0.0	0.1	0.2	50.4	
22	ACS Liquid Mercury ST (Concentrated) (liquid)[c]	0.25	0.25	0.25	0.75	0.0	0.0	0.1	59.0	
23	ACS Liquid Mercury ST (liquid)	0.75	0.75	0.75	1.50	0.1	0.1	0.8	50.4	
24	Fairview Liquid Mercury SD (liquid)	0.75	0.75	0.75	2.00	0.1	0.1	0.1	48.2	
25	Niadual (liquid)	2.00	1.40	1.40	5.00	0.1	2.7	2.1	55.9	
26	New Gallotox (liquid)	0.75	0.75	0.75	1.50	0.3	1.0	1.1	54.8	
27	Canuck Liquid Mercury (liquid)	.75	.75	.75	2.25	0.2	1.2	0.2	51.2	
28	Memmi (liquid)[c]	(.75 oz. of 1:7 for cereals)			(.75 oz. of 1:3 for flax)	1.8	6.9	1.1	59.8	
29	Morven (liquid)	.75	.75	.75	1.50	10.7	7.1	9.1	63.6	
	Significant difference (5%)					3.08	0.80	3.88	4.56	

[a] Means of 7 Stations for wheat, 10 Stations for oats, 11 Stations for barley, 10 Stations for flax.

[b] For treatments marked by asterisk (*) the dosage was based on the following seeding rates: 1.25 bu./acre for wheat, 1.75 bu./acre for oats and barley, and 0.50 bu./acre for flax.

[c] Diluted with water before use.

EFFECTS OF HIGH SURFACE-SOIL TEMPERATURE ON CEREALS AND FLAX[1]

T.C. Vanterpool[2]

Abstract

The effects of high surface-soil temperatures on cereals and flax are reviewed. On cereals, the effects may be of the leaf-banding type, the basal heat-canker type or a lodging and breaking-over type following inadequate crown-root development. On flax, there is early seedling blight and killing, and a breaking-over of older plants resulting from a constriction at ground level. Measurements with thermocouples showed that on clear days when the air temperature was 96°F (35.5°C), surface-soil temperatures on bare areas between the rows in wheat fields and on summer fallow ranged from 134° to 139°F (56.5 to 59.5°C).

Heat damage of various kinds to cereal and flax crops is of common occurrence on the Canadian Prairies. The particular type of damage usually depends on the stage of plant development and is also closely associated with other environmental conditions, such as drought. Most enquiries about these troubles are received from growers early in the season. This suggests that the growers do not expect heavy damage from heat so early in the year and are inclined to attribute the troubles to other causes. The injury is, in fact, not caused by the direct action of the sun's rays on the young plant tissue, but by the heating of the dry soil surface by strong insolation on the afternoons of hot, calm days. Similar injury by freezing on near-freezing temperatures was reported earlier (1).

Injury to cereal seedlings is of the leaf-banding type (Fig. 1). This condition is also illustrated in an earlier paper by the author (Vanterpool, 1949); slightly older plants may show the heat canker or basal constriction type of injury (Fig. 2); and plants well past the crown-rooting stage may show a lodging or breaking of the sub-crown internode caused by the failure of the crown roots to develop in the hot, dry, surface soil (Fig. 3). These latter conditions were also illustrated earlier (Vanterpool, 1959). Injuries to flax caused by high surface-soil temperatures may be of two kinds, namely: an early heat-canker or seedling blight (Fig. 4); or a lodging or breaking-over of older plants in which the damaged zone shows as a constriction at ground level (Fig. 5).

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[1] Contribution from the Department of Biology, University of Saskatchewan, Saskatoon, Sask., with financial assistance from the National Research Council, Ottawa.

[2] Professor of Biology (Plant Pathology).

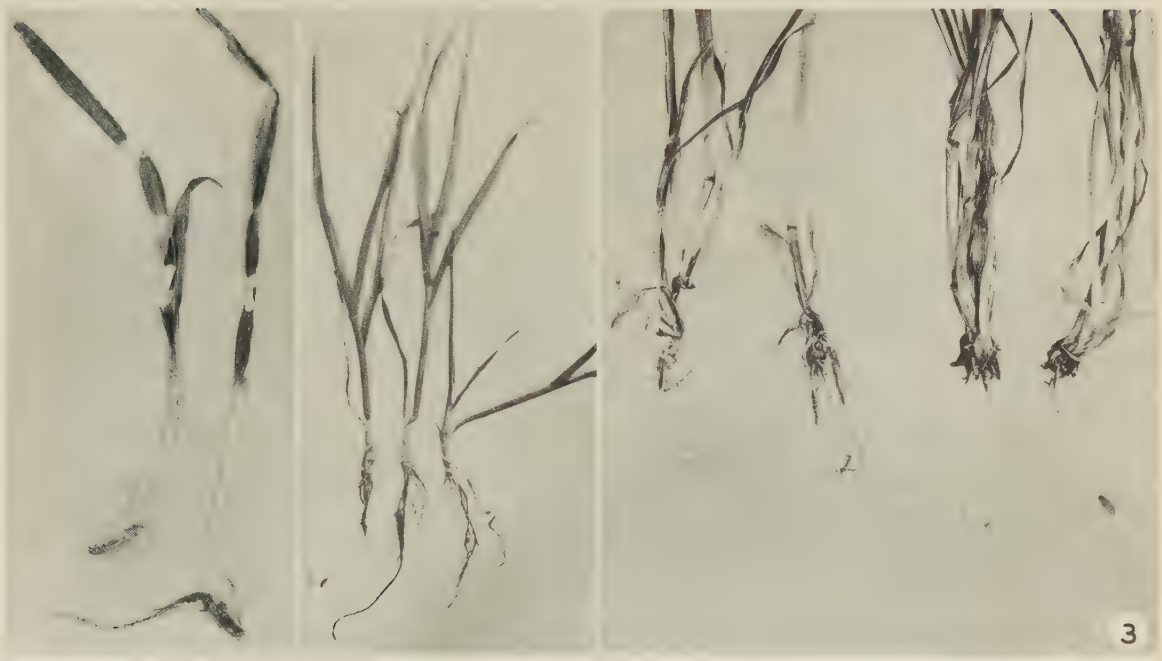

Figure 1. Leaf banding of oats caused by high surface-soil temperature.

Figure 2. Heat canker and basal contribution on oats.

Figure 3. Failure of crown roots of oat plants to develop.

Damage of the heat-canker and leaf-banding types on flax and cereals in 1961 was the most serious and widespread ever encountered in Saskatchewan. Exceptionally favorable to this trouble was the dry, hot, clear weather with sub-normal wind velocities that began during the last week in May and continued for virtually the rest of the summer. The only appreciable break was on June 12 and 13 when 0.64 inches of rain fell. The period from June 3 to 7 (Table 1) was most favorable for heat damage to young crops. No dew deposits were recorded and immediately afterward numerous reports of general leaf banding of cereals were received from Radville and Estevan in southern Saskatchewan to as far north as Debden. Heat canker on seedling flax was also general and about two weeks later the breaking-over type of injury was very damaging in central Saskatchewan with the most reports coming from Kindersley and the area stretching eastward through Rosetown and Saskatoon to Meacham.

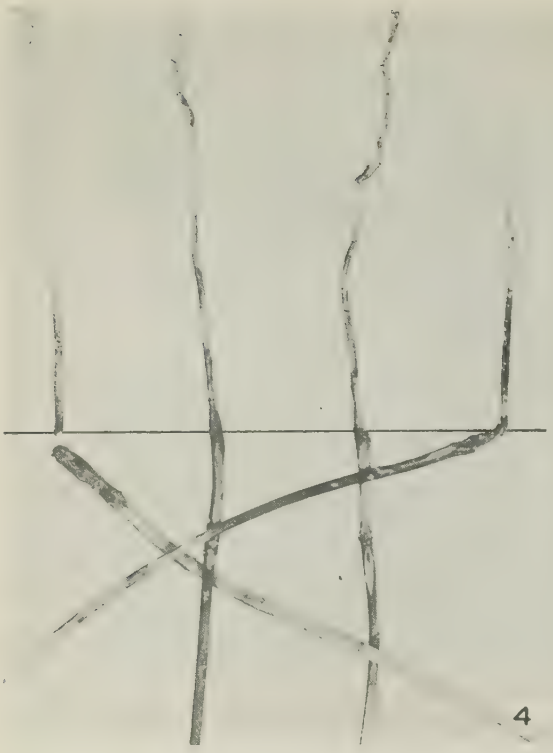

Figure 4. Heat canker seedling blight of flax.

Figure 5. Heat canker – late type on flax.

Table 1. Some meteorological records at Saskatoon
University of Saskatchewan, June 3 to 7, 1961

Date June	Afternoon air temp. °F.	Minimum night temp. °F.	Total hours sunshine	Wind velocity
3	92	57	15.7	10.1
4	95	57	13.2	6.2
5	94	60	14.5	10.7
6	96	65	15.8	10.1
7	95	63	12.3	8.5

The surface-soil temperature records reported earlier (1) were taken with laboratory thermometers and lack the precision obtained with thermocouples. In the studies reported here, special instruments with delicate thermocopule measuring points were loaned by the Agricultural Engineering

Department, University of Saskatchewan and the Building Research Department, National Research Council. On June 6, when the air temperature was 96°F, soil-surface temperature readings were taken between 1.30 and 2.30 p.m. in areas between rows in wheat fields and on bare summer fallow receiving direct insolation. The highest surface-soil temperatures recorded ranged from 134° to 139°F; some 10 to 15°F higher than the minimum required for injury to plant tissue. Soil color and degree of compaction are factors responsible for some of the temperature variations.

As pointed out in an earlier paper (1), leaf-banding of cereals with consequent "flagging" of damaged leaves in the wind delays growth only slightly and does not cause appreciable thinning of the crop unless the internal growing points happen to be emerging through the soil-surface on hot afternoons. Seedlings with injured growing points are likely to be killed. Stands of flax, on the other hand, are frequently liable to severe thinning. Many young seedlings may be killed and on many others a constriction may form at ground level. These partially damaged young plants are later broken over by strong winds, further reducing the stands. In 1961, many flax fields near Rosetown had 15 to 20 per cent of this late type of heat canker. They were so thinned that they were unable to adequately compete with weeds and, in some instances, were plowed under.

The effects of high surface-soil temperature referred to here can be alleviated by sowing early and at slightly higher rates than normal. Soil packing to encourage even germination, shading provided by surface trash, and seeding in a north-south direction are also beneficial.

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FORECASTING LATE BLIGHT OF POTATO IN THE MONTREAL AREA IN 1961

Thomas Simard[1]

Abstract

According to the results obtained at 6 localities in southwestern Quebec, the methods of forecasting developed by Hyre and Wallin again reflected quite well the potato blight situation in that area during the 1961 growing season. As recorded by the two methods, many blight - favorable periods occurred at all stations and an epidemic was predicted. At the end of July, the 95 per cent point of foliage destruction was observed in fields of early varieties and at the end of August, in fields of late varieties. Under the conditions that prevail in southwestern Quebec, it seems that blight-favorable periods occurring before the last week of June are valid for forecasting the first appearance of blight on early varieties but that they might possibly be ignored in forecasting the disease on late varieties.

Introduction

Promising results were obtained in 1960 with two methods of forecasting late blight of potato (4). These investigations were continued in 1961 with the aim of eventually establishing a Spray Warning Service for the potato growers of the Montreal area.

Methods and Procedure

The main method used was the moving-graph or rainfall-temperature method devised by Hyre (2). A modification was brought to this method because of the fact that, under local conditions, the 5-day average temperature for the growing season seems always to be below 78°F. (5). The temperature, therefore, is always considered to be favorable for late blight except when the minimum is less than 45°F. Rainfall is considered favorable when the 10-day total is 1.20 inches or more. Late blight is expected to first occur 7 to 14 days following a period of 10 consecutive days when both temperature and rainfall are favorable, provided that the weather continues to be favorable for blight. The count of consecutive favorable days is not interrupted by an unfavorable day due to low temperature; that day is simply omitted from the count. Once the disease is established, 10 favorable days are no longer required for its spread. A degree of flexibility is desirable in applying these criteria.

The second method investigated was the 90 per cent relative humidity-temperature method developed by Wallin (2, 3). With this method, hygrothermographs, activated at the time of potato emergence, are located 12 to

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15 inches above ground in shelters placed near potato fields. Severity values for late blight infection are calculated as indicated in Table 1.

Table 1. The relation of the hours duration of given average temperature coincident with relative humidities of 90% or more to the estimated severity of secondary infections of Phytophthora infestans.

Average temperature range, °F.	Hours that produce indicated infection severity				
	1	2	3	4	8
	Trace	Slight	Moderate	Heavy	
45-53	16-18	19-21	22-24	25-29	30-
54-59	13-15	16-18	19-21	22-26	27-
60-80	10-12	13-15	16-18	19-23	24-

Interpretation of cumulated severity values

20 - zero time. First spray
3 per week - Additional spray
1 per week - The blight fungus stays alive
0 for 3 or more consecutive weeks - The fungus cannot survive.

The two methods were used in the three following districts on the muck soil area south of Montreal: Ste. Clothilde, Napierville (St. Blaise) and Farnham (Ste. Sabine). Hyre's method alone was used at Lennoxville (east of Montreal), and Duvernay and St. Thomas (north of Montreal). The results of this study appear in Figure 1.

Results

The forecasting methods investigated gave again this year a good picture of late blight development. It can be seen that, in all districts, numerous blight-favorable periods were recorded by the two methods. Accordingly, growers were advised early not to neglect the spraying of their fields since a blight epidemic was indicated. In fact, at the end of July the 95 percent point of foliage destruction was observed in fields of early varieties and at the end of August, in fields of late varieties. In almost all localities, blight was first observed on late varieties about one month after it was forecast. As mentioned before (4), this might be due to the difficulty of detecting very early symptoms.

Another explanation might be that early forecasts are more valid for early than for late potato varieties. In England (1), flushes of blight-favorable periods that occur before the last week of June are not considered for forecasting blight on late varieties. The same situation might prevail under local conditions. In the Ste. Clothilde-Napierville district, the 95 percent point of foliage destruction was observed in Irish Cobbler fields at the end of July, indicating that blight must have established itself early in July in accordance with the forecast made using Hyre's criteria.

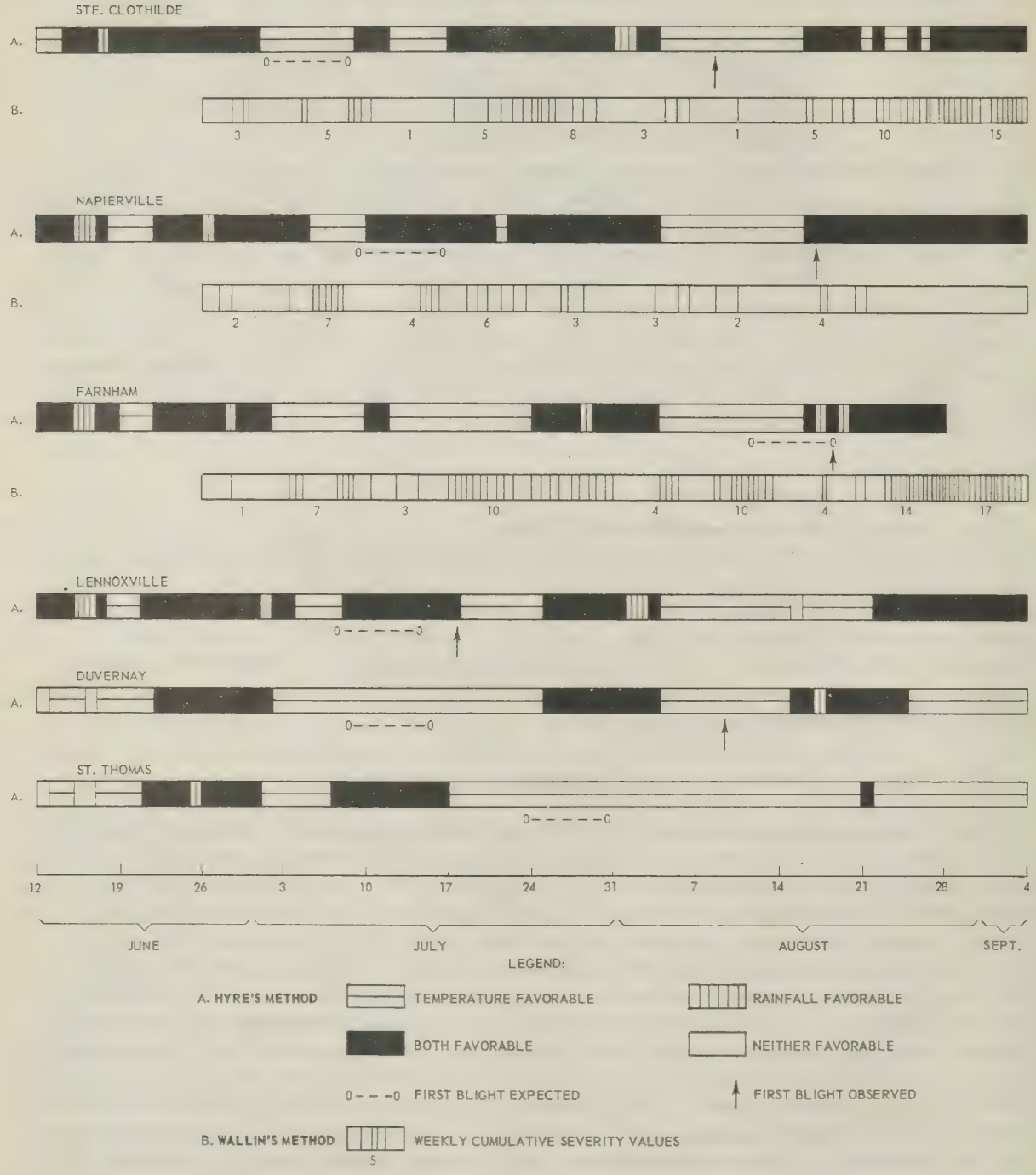

FIGURE 1. FORECASTING LATE BLIGHT

This seems to agree with the results obtained by Wallin's method at the localities where it was used. As noticed in Figure 1, the hygro-thermograph data were only available on June 26. The zero point (cumulated severity values of 20) occurred during the last week of July, that is, a few days before blight was observed in fields of late varieties. The results of investigations on spraying potatoes according to the two forecasting methods (6) seem also to agree with this conclusion.

Acknowledgements

I wish to thank the following persons for their kind collaboration: Mr. J.J. Jasmin, Officer-in-Charge, and the personnel of the Substation for organic soil of Ste. Clothilde; Mr. M. Volk (Hardee Farms Ltd.); Mr. William Max (Farnham Farms, Ltd.); the personnel of the Experimental Farm at Lennoxville and of the Quebec Plant Protection Stations at Ste. Clothilde, Duvernay and St. Thomas.

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SPRAYING POTATOES ACCORDING TO TWO METHODS OF FORECASTING LATE BLIGHT

Thomas Simard and Jacques Simard[1]

Abstract

Late blight on foliage was effectively controlled by 7, 5 and 9 sprays applied, respectively, according to Hyre's and Wallin's methods of blight forecasting and to normal farm practice. Sprays applied before July 26 were of no value. It appeared that the first appearance of blight on late varieties was more accurately forecast when favorable periods occurring before the last week of June were disregarded. The data indicated that the yield was substantially decreased in unsprayed plots.

Introduction

Under the conditions prevailing in southwestern Quebec, late blight of potato varies in its development and intensity from year to year. In order to obtain the best control with a minimum of fungicide sprays, it is important to be able to predict when and how the disease will occur and to spray accordingly. This involves the use of a method or methods for predicting late blight infection periods for proper timing of fungicide applications. This paper is a report of the initial year of an experiment dealing with this problem.

Methods and Procedure

This experiment was carried out on muck soil at the Canada Agriculture Substation at Ste. Clothilde. It included 12 plots of the late variety Green Mountain, randomized in 3 replicates. The plots, consisting of 8 rows, were 50 feet long and bordered by 2 unsprayed rows.

The two methods of late blight forecasting used in this experiment were those developed by Hyre and Wallin (1). Hyre's method was modified slightly regarding the temperature criteria, as explained in (1). The recording of the data for Hyre's method started on potato emergence, on June 12. Those for Wallin's method two weeks later, on June 26. The treatments were as follows:

A - Spraying according to Hyre's method of forecasting
B - Spraying according to Wallin's method of forecasting.
C - Spraying according to normal farm practice.
D - Unsprayed.

The fungicide used was Maneb 50-W, 2 lb. per acre, except for one application of a copper dust in treatment C. Due to soil conditions, the fungicide applications were usually made by hand with a spray gun. Otherwise, a high power potato sprayer was used. The tops were chemically killed on September 5, two weeks before harvest. Yield records were taken in the 4

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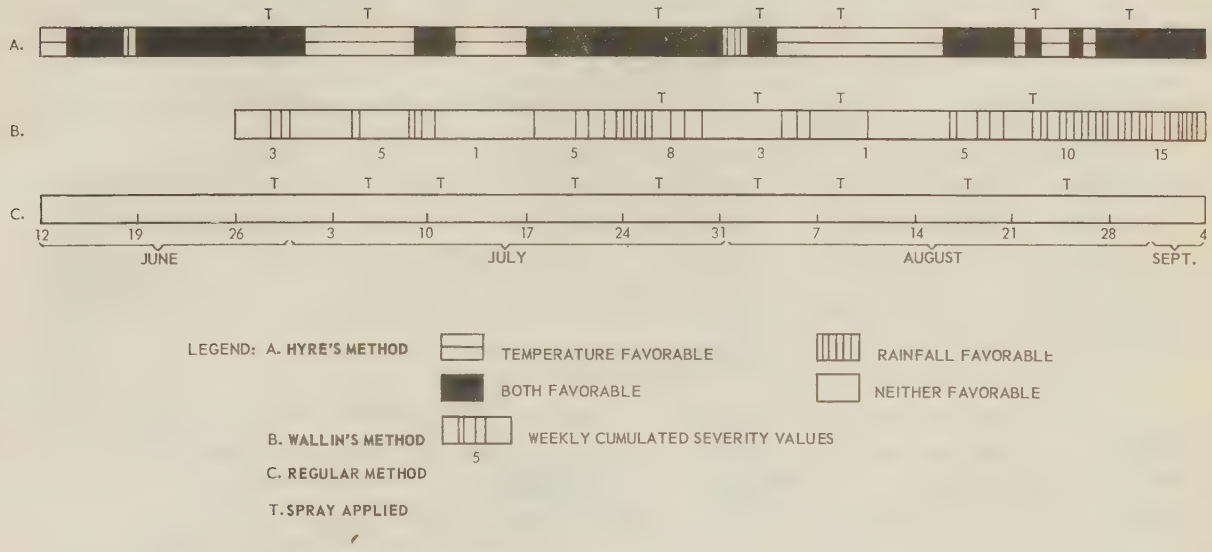

FIGURE 1. SPRAYING POTATOES IN ACCORDANCE WITH TWO METHODS OF FORECASTING LATE BLIGHT

centre rows of each plot. A summary of blight infection periods and fungicide applications are given in Figure 1.

Results

Figure 1 shows that 7 treatments were applied according to Hyre's method, 5 according to that of Wallin and 9, according to normal farm practice.

With respect to blight on the foliage, there was no difference between treatments A, B and C, there being only traces of the disease in all three treatments at the end of August. Readings in check plots were as follows: August 9, about 10%; August 24, about 40%; August 28, 95%.

These results show that sprays applied before July 26 were not of value and that Wallin's method was more accurate for the forecasting of the first appearance of blight. This is in agreement with the results of another study (1) which indicated that blight-favorable periods occurring before the last week of June might be disregarded for forecasting the disease on late varieties. The yield records are given in Table 1.

Due to the nature of the land used (floodings, damage by machinery), the figures in Table 1 are not statistically significant. It is obvious, however, that the yield in unsprayed plots was substantially decreased by late blight.

Table 1 - Yield in pounds of sound No. 1 marketable potatoes

Treatment	Replicate 1	2	3	Total
A	173	238	393	804
B	246	397	188	831
C	69	381	397	1073
D	57	137	94	288

Acknowledgements

We express our thanks for their kind collaboration to Mr. J.J. Jasmin, Officer-in-Charge, and the personnel of the Canada Agriculture Substation at Ste. Clothilde, and to K. Choquette, P. Lavigne, R. Garneau and A. Paris, Information and Research Service, Quebec Department of Agriculture.

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FAIRY RINGS IN ALBERTA[1]

J.B. Lebeau[2] and E.J. Hawn[3]

Introduction

The incidence of fairy ring reached a peak in 1961 making it the most important turf grass disease in southern Alberta. The fungus was so destructive that in some cases entire lawns had to be torn up and resodded with healthy turf or reseeded. Park lawns and golf course fairways were damaged or became unsightly by the development of the disease and even farmers and ranchers expressed concern over the appearance of the rings on native grass pastures.

The disease was described more than a century ago but very little is understood about the host-parasite relationships and there are, as yet, no satisfactory control measures (4).

Symptoms

The first evidence of the disease in Alberta is usually continuous or interrupted circular bands of green grass (Fig. 1a). Fruiting bodies may appear in the rings (Fig. 1c and d) from time to time under conditions of sufficient moisture. Later the affected grass often wilts, turns brown, and dies and the ring is often invaded by weeds. Invariably the soil under the dead portion of the ring is compact with mycelium and often so hard that mechanical aerators cannot penetrate it. The size of the ring will vary, depending on age, from less than one to over 200 years in diameter.

Etiology

The fungi associated with fairy rings have been identified and reported in the literature. Shantz and Piemeisel (5), in addition to their own investigations, report on the work of 31 authors dealing with 47 fleshy fungi. We were unable to obtain any fruiting bodies of the fungus causing the large rings illustrated in Fig. 1 but Marasmius oreades Fr. was identified as the causal organism of the rings reported from the other districts in southern Alberta.

Many attempts have been made to determine the nature of pathogenesis in this disease but no general agreement has been reached. Bayliss-Elliott (2) considers that the death of the grass is due to parasitic action of the fungus and disagrees with Shantz and Piemeisel (5) who believe that the death of the grass is caused by drought produced by compact growth of the fungus in the soil. Biochemical processes that take place in the soil during the progress of the fairy ring fungi have been investigated but have not thrown much light on the host-parasite relationship. The conclusion drawn from these studies (3) is that the protein portion of the organic matter in the soil is converted by

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[2] Head, Plant Pathology Section.
[3] Plant Pathologist.

the fungus into compounds of nitrogen that are readily available to higher plants. The largest amounts of free ammonia were found in the ring area and less without than within the ring. No attempts, however, were made to show that any of these chemical reactions has a lethal effect on the host. Although Bayliss-Elliott (2) states that there are indications of a toxic excretion produced by the fungus that damages the roots of the grass and enables the fungus to attack them even before they are dead, she offers no substantial evidence to support his hypothesis. Albrecht and Sheldon (1) showed that grass produced from fairy rings caused by M. oreades contained twice as much protein as the non-ring grass and that all the nine essential amino acids assayed were present in higher quantities in the ring grass. This lush, high protein grass is often over-grazed and may be another contributing factor to the death of grass on range land.

Epidemiology

The tendency of fungi to grow outward from the point of germination of the spore or from the original source of infection results in circular colonies. By this procedure the size of the fairy ring increases from year to year. Bayliss-Elliott (2) found the maximum increase of a ring produced by M. oreades to be 13.5 inches per year. From this the age of the large rings on range land in southern Alberta (Fig. 1f) may be estimated to be over 200 years old. Presumably new rings are initiated from the point of germination of the fungus spore although Shantz and Piemeisel (5) claim that only on rare occasions are conditions favorable on the Great Plains for spore germination of basidiomycetes. It is quite possible that the rings are often produced from fragments of very old ones.

Distribution

This distribution of fairy rings in southern Alberta is widespread and has been reported on lawns, golf courses, parks, pastures, and range land. Typical symptoms on these crops from representative districts are illustrated in Fig. 1.

Fairy rings appear chiefly in grassed soils that are dry and low in fertility. This statement is often disputed by home owners who claim they have supplied their lawns with sufficient amounts of fertilizer and water and are still plagued with fairy rings. However, the disease is prevalent on golf course fairways but seldom develops on the greens and the latter are fertilized and watered more frequently than any of our grassed areas.

Discussion

Fairy rings, regardless of their stage of development, have an undesirable effect on turf grass planted principally for ornamental and recreational purposes. These areas are costly to prepare and maintain and methods for prevention and eradication of the disease would be of practical significance.

Very little research has been done on this disease. A review of the literature reveals that most of the investigations were done prior to 1920 and no record could be found of any work done in Canada. We believe that the

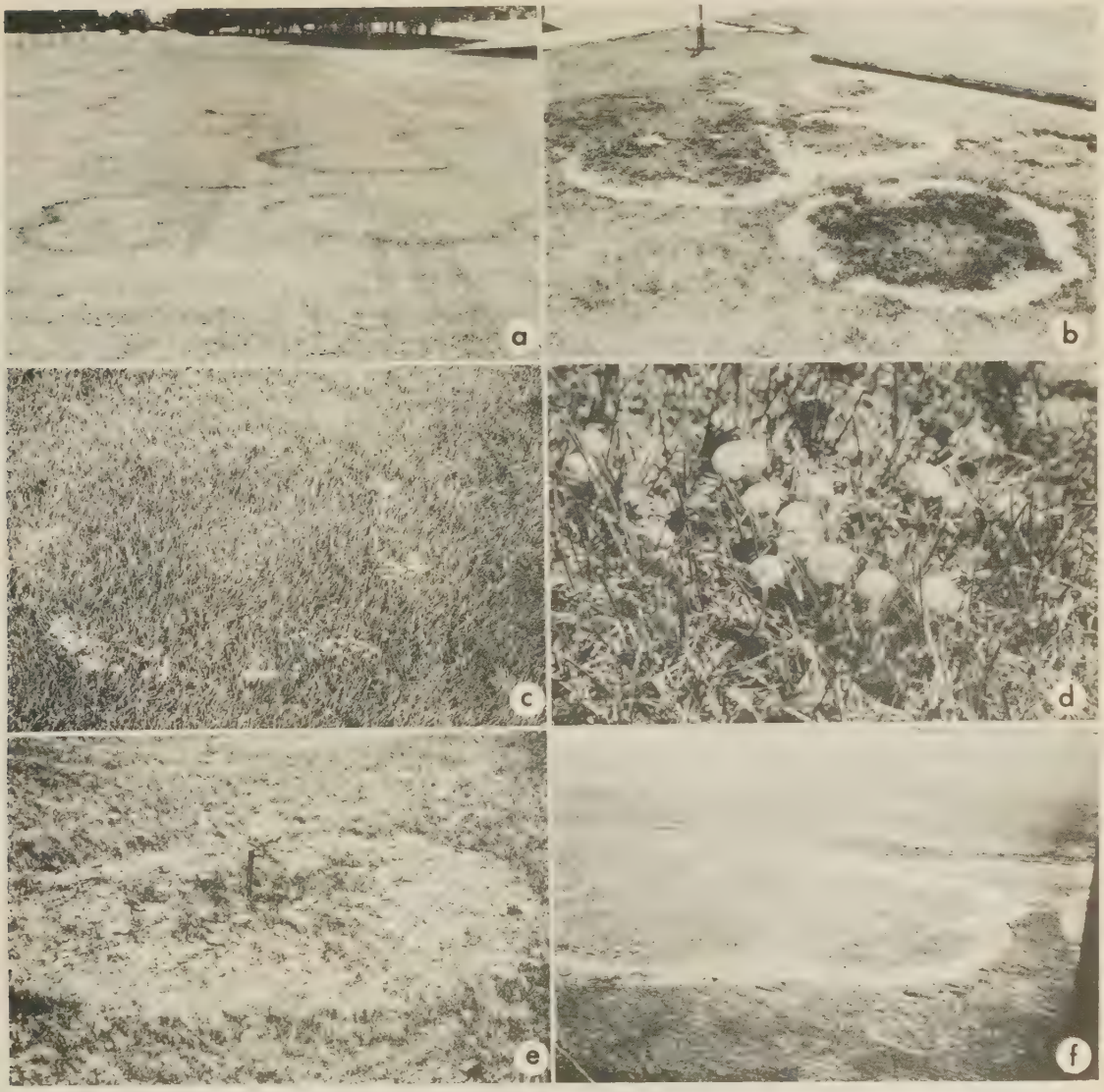

Figure 1. Fairy rings in Southern Alberta.

a) Incipient symptoms of green banding on fairway of Golf and Country Club, Calgary, Alberta.

b) Severe damage on a lawn at Lethbridge, Alberta.

c) Fruiting bodies of *M. oreades* in typical ring formation.

d) Close-up of fruiting bodies shown in Fig. 1c.

e) Damage in native pasture near Barons, Alberta.

f) Aerial photograph of range land south of Manyberries, Alberta. Large ring shown measured ½ mile in circumference.

disease warrants further investigation and have initiated a program to study the host-parasite relationship and are experimenting with several methods of control.

Acknowledgement

The authors wish to express their thanks to Mr. S. Smoliak, Agrologist at the Experimental Farm, Manyberries, for supplying them with the aerial photograph used in this paper.

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LA TACHE ARGENTEE DANS LA PROVINCE DU QUEBEC[1]

Jacquelin Santerre[2]

Résumé

Des échantillons de tubercules de pommes de terre de diverses variétés ont été examines pour déterminer la fréquence de la tache argentée dans nos champs. Ces tubercules provenaient de différentes localités comprenant un peu plus de 50% de la superficie totale de production de pommes de terre de la province du Québec. L'examen a démontré que la tache argentée, causée par Helminthosporium atrovirens (Harz) Mason et Hughes (Spondylocladium atrovirens Harz.) a été beaucoup plus fréquente en 1960 qu'une autre maladie des pommes de terre, la dartrose, causée par Colletotrichum coccodes (Wallr.) Hughes (C. atramentarium (Berk. et Br.) Taub.). Tous les tubercules examinés hébergeaient l'organisme de la tache argentée, tandis que seulement 3.2% de ceux-ci étaient atteints de dartrose.

Abstract

Samples of potato tubers of different varieties were examined in order to determine the importance of silver scurf in Québec fields. These tubers were grown in various localities representing more than 50% of the total potato acreage of the province of Québec. Silver scurf caused by Helminthosporium atrovirens (Harz) Mason & Hughes (Spondylocladium atrovirens Harz.) was found to be much more prevalent in 1960 than potato black dot caused by Colletotrichum coccodes (Wallr.) Hughes (C. atramentarium (Berk. & Br.) Taub.). All the tubers that were examined harboured the fungus of silver scurf, while only 3.2% of them were infected by black dot.

Introduction

Depuis quelques années, la tache argentée, causée par Helminthosporium atrovirens (Harz) Mason et Hughes (Spondylocladium atrovirens Harz), semble prendre de l'ampleur chaque année au point de causer des pertes considérables. Cette situation a été observée au Brésil, aux Etats-Unis et aux Pays-Bas (1, 2, 3). Récemment, on a fait les mêmes constatations dans la province de l'Ontario (7), lorsque l'examen des semences, au printemps de 1958, a montré que des tubercules de catégorie Fondation, aussi bien que des tubercules de semence certifiée étaient, dans la plupart des cas, infectés par la tache argentée, qu'elle qu'ait été leur provenance.

Dans un rapport antérieur (4) d'une enquête pour déterminer l'importance relative de la tache argentée et de la dartrose (Colletotrichum coccodes (Wallr.) Hughes) dans les champs de pommes de terre du Québec,

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[1] Contribution de la Station de Recherches, Ministère de l'Agriculture du Canada, Ste. Anne de la Pocatière, P. Qué.

[2] Phytopathologiste.

l'auteur mentionnait une prédominance marquée de la première de ces duex maladies dans les quelques centres de production où cette enquête a été poursuivie. Celle-ci fut par la suite étendue à un plus grand nombre de centres de production du Québec, afin d'obtenir une image plus exacte de l'état de la tache argentée à la suite de la récolte de 1960.

Matériel et méthodes

Grâce à la coopération des inspecteurs du Bureau de Protection des Plantes de Ste-Anne-de-la-Pocatière, il nous a été possible d'examiner quelque 767 tubercules de diverses variétés provenant de différents endroits de cette province.

Ces tubercules de semence, recueillis au hasard dans les caveaux et les entrepôts des producteurs à l'inspection printanière, provenaient des variétés suivantes: Green Mountain, Irish Cobbler, Katahdin, Kennebec, Keswick et Teton. Le territoire sur lequel a été menée cette enquête comprend un peu plus de 50% de la superficie totale de production de cette récolte dans la province.

A leur arrivée au laboratoire, les tubercules ont d'abord été brossés à l'eau courante et déposés ensuite sur un lit de vermiculite humide, suffisamment espacés dans des caissettes de bois pour éviter tout contact entre eux. Après avoir recouvert ces caissettes d'une feuille de vinyl transparent permettant de surveiller les progrès de la maladie, nous les avons conservées à 80°F dans une chambre à température constante.

Comme les tubercules soumis à l'examen présentaient différents degrés de sévérité de la maladie, depuis l'absence totale de lésions jusqu'à l'envahissement partiel de la pelure par la tache argentée, l'incubation a été maintenue assez longtemps pour permettre au champignon de fructifier sur tous les tubercules atteints et obtenir ainsi un diagnostic sûr de la maladie. La durée de l'incubation a ainsi varié de 3 à 27 jours.

Enfin tous ces tubercules ont été examinés au binoculaire pour le diagnostic final.

Résultats et discussion

Les résultats obtenus au cours de cette étude sont groupés au tableau 1.

Comme ce compte-rendu fait suite à un rapport précédent (4), sur l'importance relative de la dartrose et de la tache argentée dans la province du Québec, nous avons jugé utile d'inscrire aussi au même tableau les observations concernant la fréquence de la dartrose en regard de la fréquence de la tache argentée.

Même si au départ un grand nombre des tubercules examinés ne montraient aucun signe extérieur de la tache argentée, ces résultats indiquent cependant que tous hébergeaient l'organisme de cette maladie, sans égard à la variété et à la provenance.

En dépit de conditions atmosphériques favorables, à l'été de 1960, la dartrose a été peu fréquente (3.2%) si on la compare à la tache argentée (100%) (Tableau 1). Ces observations concordent bien avec les données rapportées ailleurs où nous avions noté des pourcentages d'infection de 100 et 6 respectivement pour la tache argentée et la dartrose.

Tableau 1. Fréquence de la tache argentée et de la dartrose (récolte de 1960)

Variété	Nombre d'échantillons	Nombre de tubercules	Pourcentage des tubercules atteints de	
			Tache argentée	Dartrose
Green Mountain	35	458	100	2.4
Irish Cobbler	2	31	100	0
Katahdin	2	25	100	8.0
Kennebec	14	140	100	4.3
Keswick	7	97	100	6.2
Teton	1	16	100	0
		moyenne =	100%	3.2%

Le fait que tous les tubercules examinés, provenant de la récolte de 1960, aient hébergé l'organisme de la tache argentée, ne signifie pas nécessairement que tous les tubercules étaient atteints de cette maladie à la récolte. En effet, puisque l'examen de ces tubercules ne s'est fait qu'au printemps, il se peut fort bien qu'un certain nombre d'entre eux se soient contaminées au cours de la période d'entreposage.

Les risques de contamination par des spores ou des fragments de mycélium d'un tubercule la l'autre au cours des manipulations nous paraissent amoindris, si on considère certaines particularités de temps qui caractérisent l'invasion des tubercules par les spores de ce champignon et le développement subséquent de la maladie. D'après Burke (1), si le mycélium et les spores peuvent engendrer des lésions, il semble toutefois beaucoup plus difficile de les obtenir artificiellement avec le mycélium suel. Par contre, avec les spores, l'invasion des tissus, après la pénétration du tube germinatif, peut prendre, d'après des observations macroscopiques, 16 jours en moyenne en chambre humide, les conidies n'apparaissant que 4 semaines en moyenne après l'invasion des tissus par le champignon. Le meilleur moment pour déceler la présence de la maladie serait donc de faire enquête au moment de l'arrachage.

Une telle enquête devrait être répétée durant un certain nombre d'années de façon à recueillir des données sur des récoltes qui auraient ainsi été soumises à diverses conditions de température et d'humidité au cours de la période de croissance. Cette étude permettrait sans aucun doute d'établir si la tache argentée n'affecte en réalité que quelques tubercules d'une plantation, comme il apparaît parfois à l'arrachage, ou si les tubercules n'en sont pas tous atteints à divers degrés. Si cette dernière hypothèse s'avérait, on pourrait alors s'interroger sur la "virginité" des soils à pommes de terre quant à cette maladie, dont la pérennité ou l'omniprésence serait le fait du saprophytisme -- les tubercules n'en étant peut-être atteints que dans des conditions déterminées.

Il ne semble faire aucun doute que l'inoculum transporté par les tubercules de semence peut transmettre cette maladie d'un sol contaminé à un sol vierge, si tant est qu'il en existe. Burke (1) et Schultz (5), pour

leur part, vont même jusqu'à prétendre que l'infection des tubercules provient en grande partie de l'inoculum apporté par le tubercule de semence contaminé. Cela est sans doute vrai jusqu'à un certain point. Rien ne répugne, cependant, à ce que le sol soit lui-même directement à l'origine de cette maladie, comme le laissent entrevoir les études de Taubenhaus (6). Les tubercules de semence porteurs de l'organisme, en étant plantés dans d'autres champs l'année suivante, n'aient tout simplement contaminer ces derniers s'ils ne l'étaient pas déjà, ou renforcer l'inoculum déjà présent. Dès lors, il ne serait pas surprenant qu'on n'ait pas accordé plus d'importance, dans le passé, à la tache argentée, si on s'est toujours contenté d'un simple examen visuel des tubercules à la récolte.

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THE HYPERSENSITIVE REACTION OF CERTAIN POTATO VARIETIES TO INFECTION WITH VIRUS X[1]

James Munro[2]

Introduction

A new Canadian potato variety which is field-immune to infection with potato virus X has been introduced. This variety, called Hunter, is a product of the National Potato Breeding Project of the Canada Department of Agriculture at Fredericton, N.B. Although Hunter is the first new variety to be introduced into North America with resistance of this kind to potato virus X, this so-called 'field-immunity' is common in many European varieties.

Immunity to all strains of potato virus X was obtained for the first time in the U.S.D.A. Seedling 41956 (6), and the factor for this immunity was shown to be transmissible to hybrid progeny (7). Although immunity to other important potato viruses has not yet been found, Cockerham (2) has shown that some varieties and seedlings have a marked resistance to systemic infections with viruses A and X under field conditions. This resistance is due to an extreme sensitivity and consequent rapid death of tissue around the infection points of inoculated leaves. The elimination of these two viruses from potato stocks is now being made possible by the production of new varieties possessing these genetic factors. Hunter is such a variety.

Field Immunity

Fundamental differences between Old World potato varieties may be observed in the way varieties react when infected with any one of the potato viruses X, A, B or C (2). Some develop local necrotic lesions whilst others produce non-necrotic symptoms. These reactions distinguish varieties and are based upon simple genetic differences (1). The necrotic reactions indicate that there will be virtual immunity to these viruses under natural conditions of infection. Two varieties named King Edward VII and Epicure, that have been outstandingly popular with growers and consumers in Britain for the past 50 years have this field immunity to viruses X and A. Experience with varieties of this kind has shown that the expression "field-immune" is apt.

When leaflets of a potato plant are inoculated with the virus to which that variety is field-immune, they develop necrotic local lesions (Fig. 1). This is usually the only result of inoculation, but occasionally the infection does not remain localized and the virus moves throughout the plant causing a systemic necrotic disease. When this happens, necroses develop most rapidly and most readily at or near to the growing points of the plant (Fig. 2.) Because of this unvarying tendency for growing points to be rapidly invaded when a field-immune plant is systemically infected, the reaction is known as top-necrosis.

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[1] Contribution No. 81 from the Canada Department of Agriculture Research Station, Fredericton, N.B.

[2] Plant Pathologist.

Experience has shown that most plants with this characteristic react with local lesions to natural or rubbed inoculations on occasional leaves, and to top-necrosis following a graft with an infected scion or to natural or rubbed inoculations to many leaves.

Occasional plants of the variety Hunter that have reacted with top-necrosis have been found in the field when sources of virus X have been brought into the growing crop by roguers or cultivation equipment. Tubers set by such plants may show obvious necrotic lesions or areas (Fig. 3). Eyes may become necrotic and fail to spout; those that do sprout give rise either to healthy plants or to plants that become necrotic and die before tubers are formed.

Hypersensitivity

The general term hypersensitivity is commonly applied to a necrotic reaction that follows a plant cell-virus interaction. Potato plants with field-immunity, or those which give a similar but less valuable reaction described as lethal necrosis, are called hypersensitive plants in their relations with a specific virus. The two reactions are superficially similar enough to be confused. Lethal necrosis is caused by the rugose mosaic virus Y in certain varieties by specific strains. Field-immunity is caused irrespective of strain by any one of the viruses X, A, B or C, to which the variety is field-immune.

A visible difference between these two kinds of hypersensitive reaction is indicated by the type of cells which show initial necrotic symptoms in the stems of systemically infected plants. In the lethal-necrosis reaction necrosis begins in the collenchyma, continues into the rest of the cortex and eventually the phloem tissue. Finally most cells from the epidermis to the pith are necrotic (4, 5). In the case of field-immune plants the cells primarily affected are in the vascular tissues.

The reaction to virus infection by lethal necrosis is valuable in that although plants in a field crop may easily become infected, the infected plants are quickly killed and do not remain a focus of infection. If tubers are large enough to be harvested they will produce, in the following year, plants that will die rapidly from lethal necrosis when they are only a few inches high.

Field-immunity is a stage ahead of lethal-necrosis in that infection usually remains localized in a leaflet, and when as occasionally happens, a plant becomes systemically infected, the necrotic tubers are glaringly obvious.

Discussion

The advantages of breeding for hypersensitivity to virus X over that of breeding for immunity (3) is that there is a linkage between the genes responsible for hypersensitivity to virus A, the cause of mild mosaic, and those responsible for hypersensitivity to virus X. Seedlings that are top-necrotic to virus X are top-necrotic to virus A, but not vice-versa. Consequently Hunter is also field-immune to virus A. There is also a wide range of possible parents that are hypersensitive to potato virus X carrying many different combinations of other desirable qualities required in a potato. Breeding for the immunity to virus X obtained from U.S.D.A. Seedling 41956 is restricted to the use of that seedling and its derivations as one parent.

1

2

3

Figure 1. Terminal and first primary leaflets on a Hunter plant inoculated with a mixture of virus X strains.

Figure 2. Hunter plant with top-necrosis of each growing point caused by virus X infected scions.

Figure 3. Necrotic lesions on tuber progeny of virus X infected plant.

The main possible weakness in hypersensitive seedlings is that of being extremely susceptible to the virus. When top-necrosis of a potato plant caused by virus X is seen in the field for the first time, there is usually doubt both as to its cause and to its relative importance. There should be no concern because the phenomenon is uncommon, it is self-eliminating, and it is abnormally conspicuous in relation to the actual number of such plants that would be in a growing crop.

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LA ROUILLE DU POMMIER[1]

J.B. Julien[2]

Une enquête a été effectuée cet été à la Station Expérimentale de Smithfield, Ont., sur la présence de la rouille du pommier (Gymnosporangium juniperi-virginianae Schw.). Elle a porté sur l'examen de 2,541 pommiers provenants de croisements de variétées et de sélections effectués depuis plus de dix ans. Ces arbres avaient été sélectionnés en serre pour leur résistance à la tavelure. Ils ont été plantés en champs et n'ont jamais été protégés par les fongicides.

Les catégories suivants ont été établi afin de déterminer la susceptibilité relative de ces arbres.

I. aucun symptomes visibles
II. présence de tachetures, absence de pycnides ou d'écidies
III. présence de pycnides, absence d'écidies
IV. présence d'écidies

Le tableau suivant montre les résultats préliminaires de cette enquête.

Tableau 1. Susceptibilité relative de plantules de pommiers à la rouille.

Parent (1)	Categorie I	II	III	IV(2)	Total	Pourcentage
Lawfam	198	00	1	0	199	1
McIntosh	739	23	260	43	1,065	30
Melba	438	89	286	50	863	49
Newtosh	59	0	1	0	60	2
tous les autres (3)	209	15	101	29	354	41
Total	1,643	127	649	122	2,541	35

(1) Géniteur commun à plusieurs croisements.
(2) Tous les écidies vus ont été identifié comme étant G. juniperi-virginianae.
(3) Tous les géniteurs non mentionnés plus haut.

Seuls les arbres de la catégorie I sont considérés ici comme résistants. Ainsi ce tableau montre que 35 pourcent des plantes sélectionnées pour leur resistance à la tavelure sont susceptibles à la rouille.

Il est à notre que les groupes d'arbres indiqués dans la première colonne montrent des variations remarquables dans leur susceptibilité à la rouille. Une analyse détaillée de ces variations permettra d'en tirer des conclusions pratiques pour l'améliorateur de plantes.

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THE CAUSES AND DISTRIBUTION OF MOSAIC DISEASES OF WHEAT IN CANADA IN 1961[1]

J.T. Slykhuis[2]

Abstract

Wheat striate mosaic, transmitted by the leafhopper, Endria inimica Say, was found on a trace to 1 percent of the plants in nearly all wheat fields examined along a route from Carlyle, Saskatchewan to Winnipeg, Manitoba. Ramsay, Stewart and Selkirk were among the most susceptible varieties tested at Ottawa. Winter wheat varieties grown in Ontario did not develop clear striate symptoms.

Another disease, possibly of virus origin, caused chlorosis, chlorotic leaf mottling, blotches and streaks, severe stunting and premature death of durum and hard red spring wheat. The disease was found in nearly all wheat fields examined on a route across southern Saskatchewan and Manitoba and affected a trace to 5 percent of the plants in different fields.

Agropyron mosaic, which has been recognized in Ontario since 1957, was first identified in a plot of spring-sown winter wheat in southeastern Saskatchewan in August, 1960. It was found in spring wheat in the same area in July, 1961.

A soil-borne mosaic was associated with a severe bronzing of winter wheat in early May in most of the districts in Ontario that lie north, west and southwest of Toronto. Although the mosaic symptoms were still evident in early June, the plants were not noticeably stunted. The leaf symptoms of the disease in Ontario differ from those of the soil-borne wheat mosaic in Illinois, U.S.A. It has not been induced by manual transmission, and no virus particles have been detected with the electron microscope. The nematicide Telone eliminated the infectivity of soil.

Wheat streak mosaic was absent or occurred on less than 1 percent of the plants in winter wheat crops examined in southern Alberta in 1961. Although the vector, Aceria tulipae K., has been found in southeastern Saskatchewan and in Ontario, the virus is known in Canada only in those districts in southern Alberta and southwestern Saskatchewan where winter wheat is grown.

A virus disease similar to, but much milder than wheat spot mosaic in Alberta, was found associated with A. tulipae on wheat at Ottawa.

Introduction

Mosaic diseases of wheat and other cereals have been recognized in Canada only in the last decade. In 1961, Hagborg (2) verified that "false stripe", a disease observed on barley in Canada since 1925, was caused by a seed-borne virus now designated "barley stripe mosaic virus". In 1952, wheat streak mosaic and wheat spot mosaic were found to be caused by viruses

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transmitted by an eriophyid mite, Aceria tulipae K. in southern Alberta (6). In 1957, Agropyron mosaic, and a mosaic disease believed to be caused by a soil-borne virus, was found on wheat in Ontario (9, 13). Although symptoms resembling wheat striate mosaic were observed on a timothy plant collected at Ottawa in 1958, and similar symptoms were found on Cornell wheat in experimental plots in 1959, transmission tests using Kent wheat as a test plant were inconclusive (8).

During 1961, wheat crops were examined for virus diseases in southwestern Ontario, eastern Ontario and the Ottawa valley. In addition, a survey was made across the southern prairies, mostly in company with Dr. W.A.F. Hagborg, Canada Agriculture Research Station, Winnipeg. We were accompanied by J.S. Horricks and Dr. T.G. Atkinson, Canada Agriculture Research Station, Lethbridge, during part of the surveys in southern Alberta. The surveys, along with experiments done during the 1960-61 season, yielded new information on the causes, distribution and possible importance of mosaic diseases of wheat in Ontario and on the Canadian prairies.

Wheat Striate Mosaic in Saskatchewan and Manitoba

Probably the most significant result of surveys in 1961 was the discovery of wheat striate mosaic in southern Saskatchewan and Manitoba. This disease was first recognized in South Dakota in 1951 (5), and has been reported more recently in North Dakota (17). Although similar viruses with different vectors are now recognized in other countries (11), only one vector, the leafhopper Endria inimica Say, is known for the wheat striate mosaic virus in North America.

Stunted plants, with fine, light-green to yellow dashes and streaks on young leaves, and severe chlorosis and necrosis on older leaves, (Fig. 1) were found in fields of Ramsay durum and Selkirk spring wheat on the farm of H. Slykhuis, Carlyle, Saskatchewan on July 1, 1961. The disease was subsequently found in about 10 other wheat fields examined in the district. About 20 additional wheat fields were examined on a survey from Carlyle through Brandon, Manitoba to Winnipeg, and the disease was found in all fields except one advanced crop in which the plants were fully headed and the leaves drying from drought. Despite the widespread occurrence of the disease, no crop was found in which striate symptoms were evident on more than 1 percent of the plants.

Diseased plants and live leafhoppers (E. inimica) were collected in southeastern Saskatchewan and taken to Ottawa. Diseased plants, collected at Winnipeg later in July by Dr. W.A.F. Hagborg, were also forwarded to Ottawa. Striate symptoms, similar to those observed in the field, developed on Ramsay durum and Selkirk spring wheat on which E. inimica, that had fed on diseased wheat for one week, were allowed to feed. An incubation period of one to three weeks was required between infection and the development of symptoms on test plants.

For further experiments, leafhoppers were collected from lawns and on grass strips between experimental plots on the Central Experimental Farm, Ottawa. E. inimica from the Ottawa area became infective after feeding on diseased wheat and were used to test the reactions of a number of varieties of durum, hard red spring, and winter wheats. It is of particular interest to note that Selkirk spring wheat and Ramsay and Stewart durum wheat, which

have been widely grown on the prairies in recent years, were readily infected with the disease and became severely stunted and necrotic. (Fig. 2). Marquis (Fig. 3) and Acadia were slightly less severely stunted. The winter wheat variety Minter, which was the main variety used in tests with wheat striate mosaic in South Dakota in 1951 (5), proved to be the most susceptible of the winter wheat varieties tested. Nebred and Winalta were also highly susceptible. Kharkov 22 M.C. (Fig. 4) developed mild striate symptoms but was not significantly stunted. None of the Ontario-grown winter wheat varieties, including Richmond, Rideau, Genesee, Cornell and Kent developed definite symptoms. Although there is evidence that wheat striate mosiac occurs in Ontario, it is not likely to become apparent on the varieties of winter wheat being grown at present.

To determine the effects of wheat striate mosaic on the growth of wheat, selected plants showing early striate symptoms, and adjacent healthy plants of comparable size were marked by tall stakes in fields of Ramsay durum and Selkirk spring wheats near Carlyle, Saskatchewan on July 3, 1961. The plants were in the jointing to early boot stage. The co-operating farmer measured the plants at weekly intervals and noted the condition of each plant. The season was unusually dry, hence growth was less than would normally be expected for the location. The results for pairs of plants on which satisfactory series of measurements were completed show that most of the diseased plants were severely stunted both in height and head development (Table 1). In addition, the plants died prematurely and there appeared to be poor kernel development.

Table 1. Heights of striate-diseased and adjacent healthy wheat plants measured at weekly intervals during July, 1961, near Carlyle, Sask.

Condition of plants on July 3	Height in inches on July 3	July 10*	July 17	July 24	Heads
Ramsay durum					
Faint striate	12	17	19	19	small
Healthy	12	18	24	30	full
Mild striate	11	15	17	17	small
Healthy	14	20	26	32	full
Mild striate	10	11	16	16	small
Healthy	11	19	24	30	full
Moderate striate	10	12	16	16	small
Healthy	13	17	24	30	full
Severe striate	10	13	16	16	small
Healthy	11	13	24	30	full
Selkirk spring wheat					
Faint striate	12	13	12	12**	small
Healthy	12	20	21	21	full
Moderate striate	13	20	21	21	moderate
Healthy	13	23	23	23	full

* Plants heading ** Plant dead

Figure 1. Fine chlorotic streaks caused by wheat striate mosaic virus on leaves of Sentry durum wheat.

Figure 2. Stewart durum wheat inoculated with wheat striate mosaic virus when in the 1 – 2 leaf stage (right) and not inoculated (left).

Figure 3. Marquis spring wheat inoculated with wheat striate mosaic virus when in the 1 – 2 leaf stage (right), and not inoculated (left.)

Figure 4. Kharkov 22 M. C. winter wheat inoculated with wheat striate mosaic virus when in the 1 – 2 leaf stage (right), and not inoculated (left). Fine chlorotic streaks developed on the leaves but the diseased plants were only slightly stunted.

Wheat striate mosaic appeared to be uniformly distributed along the route surveyed from Carlyle, Saskatchewan to Winnipeg, Manitoba, hence it probably occurs in a much wider area. Although only 1 percent or fewer of the plants appeared to be diseased in the wheat fields examined, the diseased plants were usually severely damaged. Since the vector, E. inimica, is plentiful in southern Saskatchewan, Manitoba and Ontario, and is known to be common in other grassland areas of Canada (1), an increase in the percentage of infective leafhoppers seems to be the only additional factor necessary to cause economically serious losses in the susceptible varieties of wheat being grown on the prairies. Indeed, Timian (17) has already reported in 1959, high levels of infection in durum and hard red spring wheat in North Dakota. It may be desirable to consider including resistance to this disease when breeding new varieties of wheat for the prairies.

An unidentified "chlorosis" of wheat in Saskatchewan and Manitoba

An unidentified disease, with some symptoms of the mosaic type, was first observed in 1961 during an examination of grain fields in southwestern Saskatchewan. A few plants that were light yellow-green in color, in contrast with the dark green of the normal wheat plants, were sparsely scattered in wheat fields near Maple Creek, Swift Current and Pense, Saskatchewan. Usually, there were blotches of more intense chlorosis on some of the leaves, and often, irregular chlorotic stripes as well as some yellow to white mottling. The chlorotic plants were somewhat stunted and were less vigorous than normal plants. A higher incidence of these symptoms was found in southeastern Saskatchewan in fields in which wheat striate mosaic was also present. Approximately 5 percent of the plants in some fields of Ramsay and Selkirk wheat near Carlyle had the blotchy chlorosis symptom. The disease also occurred in all wheat fields examined between Carlyle and Winnipeg.

The effects of the unidentified chlorosis disease on the growth of Ramsay and Selkirk wheat in the field were measured by the same procedure described previously for wheat striate mosaic. Selected diseased plants and adjacent healthy plants were marked by tall stakes driven in the soil beside them. The heights of the plants were measured by the farmer at weekly intervals. The results given in Table 2 are the averages of measurements taken of all diseased and healthy plants respectively for each variety at each date. None of the diseased plants grew appreciably after July 3, and, although heads began to emerge on some of the diseased plants, all were sterile. All diseased plants were dead before July 24 while the healthy plants were still green and had developed full heads. This disease appeared to be more destructive to Ramsay and Selkirk than wheat striate mosaic.

Chlorotic plants collected in southeastern Saskatchewan and southern Manitoba were taken to Ottawa where transmission tests were done with aphids and some grass-feeding leafhoppers. Similar diseased plants collected by Dr. R.D. Tinline near Kyle, Saskatchewan were also tested. Barley yellow dwarf virus was transmitted by Rhopalosiphum padi (L.) from some of the plants, but it does not appear that this virus causes the main symptoms described. Although attempts to transmit a virus from the diseased plants with Endria inimica and Macrosteles fascifrons failed, the symptoms on the naturally diseased plants resemble symptoms of certain diseases of cereals known to be caused by leafhopper-transmitted viruses.

Table 2. Heights of wheat plants with blotchy chlorosis symptoms and adjacent healthy plants measured at weekly intervals during July, 1961 near Carlyle, Sask.

	No. of plants measured	Average height in inches on July 3	July 10	July 17	July 24
Ramsay durum					
Diseased	6	10.0	9.0	8.5	plants dead, disintegrating
Healthy	6	11.7	17.7*	26.6	29.2; green, full heads
Selkirk					
Diseased	2	8.0	9.5	10.5	10.5; dead, sterile
Healthy	2	10.5	17.5*	20.5	20.5; green, full heads

*Plants headed.

Agropyron mosaic on wheat in Ontario and Saskatchewan

Agropyron mosaic has been observed on scattered plants in winter wheat fields in Ontario each year since 1957. In May and June, 1961, symptoms of Agropyron mosaic (Fig. 5), were again commonly found on scattered plants in winter wheat fields throughout the Ottawa valley. The highest infection observed in a farm field was 25 percent of the plants near a grass border which included a preponderance of Agropyron repens infected with Agropyron mosaic. The incidence of diseased plants in the wheat decreased with increasing distance from the border, and few diseased plants could be found 25 yards away.

An Agropyron mosaic nursery has been developed in experimental plots at Ottawa by seeding small plots of winter wheat at 2-to-3 week intervals from early June to October. There is immature wheat in the plot area at all times, and whenever the virus spreads there are young, susceptible plants nearby. Infection ratings can be most satisfactorily made during May. The infections that developed in plots of 4 rows each, replicated 4 times, sown on different dates in 1959, were as follows: June 10-(90%), July 28-(64%), August 11-(60%), August 25-(21%), September 8-(0%), September 22-(0%). The infection resulting in similar plots in 1960 were: June 3-(100%), June 10-(100%), June 28-(100%), July 18-(100%), July 29-(100%), August 20-(100%), August 31-(95%), September 2-(58%), September 8-(43%), September 19-(90%). These results show a very high rate of spread during the summer and fall of 1960, which is comparable to the rate of spread of wheat streak mosaic which occurs in Alberta when winter wheat is sown adjacent to naturally diseased wheat (12). Although mites are suspected to be the vectors of Agropyron mosaic, this hypothesis has not been proven.

The first evidence of Agropyron mosaic in Saskatchewan was found in a plot of Kent winter wheat that was sown in May, 1960 adjacent to natural grass pasture on a farm near Carlyle. When the plots were examined in August,

spring wheat in the area was ripe but the winter wheat in the plot was still green and, fortunately, not heavily infected with rust. Symptoms, thought at first to be wheat streak mosaic, were observed. Eriophyid mites, principally A. tulipae, were abundant. Diseased plants collected in the plots were tested at Ottawa, and it was established that the disease was Agropyron mosaic. During a visit to the same area on July 1, 1961, symptoms of Agropyron mosaic were found on plants in a field of Selkirk wheat on the same farm. On July 3, Agropyron mosaic was found on 75 percent of the plants within 10 to 20 feet of the edge of a field of spring wheat adjacent to a grass strip in which naturally diseased A. repens grew in abundance. Although some of the infected wheat plants appeared slightly stunted, it was not possible to estimate the probable effects on yield. Diseased wheat plants and A. repens from this area were tested at Ottawa. The virus isolated from both species was indistinguishable from the Agropyron mosaic virus normally isolated from wheat and A. repens in Ontario.

Soil-borne mosaic of wheat in Ontario

Mosaic symptoms attributable to a soil-borne agent have been observed on wheat in southwestern Ontario since 1957 (9). The leaf symptoms are a light-green to yellow mosaic including spots and short streaks (Fig. 6). Affected plants, when observed in the field, are usually not noticeably stunted, and to date no data are available on the effects of the disease on yield. Diseased plants are sometimes found scattered among healthy plants, but they usually occur in patches. Sometimes the symptoms occur on all plants in a field.

In 1961, surveys for soil-borne wheat mosaic were made on two dates, May 9 to 11 when winter wheat in most fields was in the stooling to early jointing stage, and June 6-9 when the wheat was in the boot to heading stages in the areas examined. No symptoms of the soil-borne mosaic were found east of Peterborough (Fig. 7), but westward, and southwestward as far as Essex County, the disease occurred in nearly all fields examined in counties where wheat is regularly grown as a major crop. All plants had mosaic symptoms in many fields examined in Simcoe, Huron, Lambton, Kent and Essex counties. Wheat is known to have been grown regularly in many of these fields. Little or no mosaic was found in areas where wheat is seldom grown.

The symptoms observed in early May included bronzing and necrosis of lower leaves in addition to the light-green mosaic on the younger leaves. The patches of affected plants could be located at a distance because of the bronze color not evident in mosaic-free areas. The mosaic was most common in lower, wetter areas in the fields. The high incidence of the disease in 1961 may be related to the cool, unusually wet conditions in early spring. Although plants with mosaic were obviously less vigorous than normal plants when observed in early May, they appeared surprisingly vigorous and not obviously weakened by the disease when observed in mid-June.

(a) Reactions of wheat varieties to soil-borne mosaic

The reactions of 11 varieties of winter wheat, 3 of winter barley and one of rye, when grown in diseased soil, were tested in boxes of soil at Ottawa, and in a farmer's field near Clinton, Ontario. For the test at Ottawa, soil, collected from fields in which diseased wheat had been found,

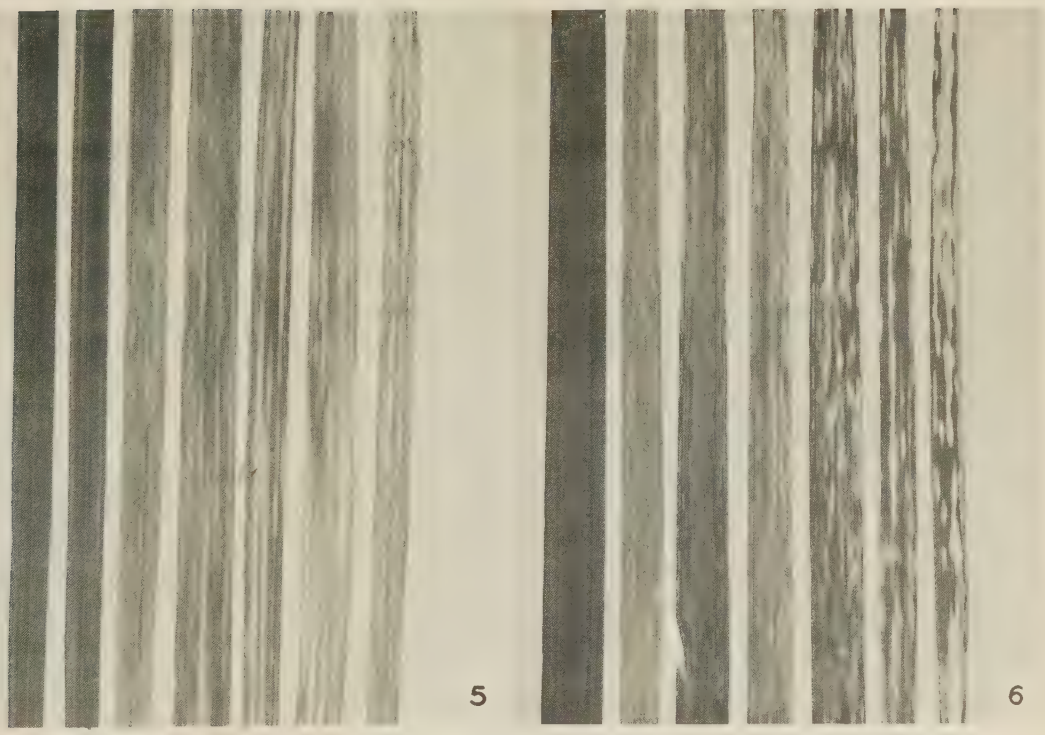


Figure 5. Symptoms of Agropyron mosaic on leaves of Kent wheat.

Figure 6. Mosaic symptoms of leaves of Genesee winter wheat grown in soil from an Ontario field in which the disease has been found.

was placed in boxes 15 3/4 x 15 3/4 x 6 inches. The varieties were sown September 20, 1960 and the boxes left outside throughout the fall and winter until March 21, 1961, when they were moved to a cool greenhouse (50°-65°F). Mosaic symptoms became evident on all varieties of winter wheat as new growth developed, and there appeared to be no major differences in reaction among the varieties, which included the following: Bison, Concho, Cornell, Dawbul, Genesee, Kent, Kharkow 22 M.C., Michigan Amber, Pawnee, Richmond and Rideau. No symptoms developed on Horton rye. The barley varieties Hudson, Kenate and Wong were winter killed, hence their reactions were not obtained. No mosaic symptoms developed on any of the varieties grown as checks in greenhouse potting soil.

The field test at Clinton, Ontario was sown September 22, 1960. When examined in May, 1961, all the wheat varieties expressed definite mosaic symptoms, but the rye and barley varieties did not.

(b) Quantities of diseased soil required for mosaic development

Tests were made to determine the quantities and mixtures of soils required for experiments to be done with greenhouse facilities. Boxes measuring 15 3/4 x 15 3/4 x 6 inches deep were filled with infective soil,

non-infective John Innes potting soil mixture, various depths of infective soil on top of greenhouse potting soil, or various mixtures of potting soil and infective soil. Four winter wheat varieties, Kent, Pawnee, Bison and Concho were sown in each box on September 20, 1960. The boxes were left outside throughout the fall and winter until March 21, 1961 when they were moved to a cool greenhouse (50°-65°F). Mosaic symptoms developed on all wheat plants grown in the following soil preparations:

(1) Infective field soil only.
(2) 3 inches of infective soil on top of 3 inches of non-infective potting soil.
(3) 1 inch of infective soil on top of 5 inches of non-infective potting soil.
(4) 1/4 inch of infective soil on top of 5 3/4 inches of non-infective potting soil.
(5) mixture of 1 part infective soil: 3 parts non-infective potting soil.
(6) mixture of 1 part infective soil: 15 parts non-infective potting soil.

No symptoms developed on plants grown in the non-infective potting soil.

(c) Elimination of mosaic infection with nematicide

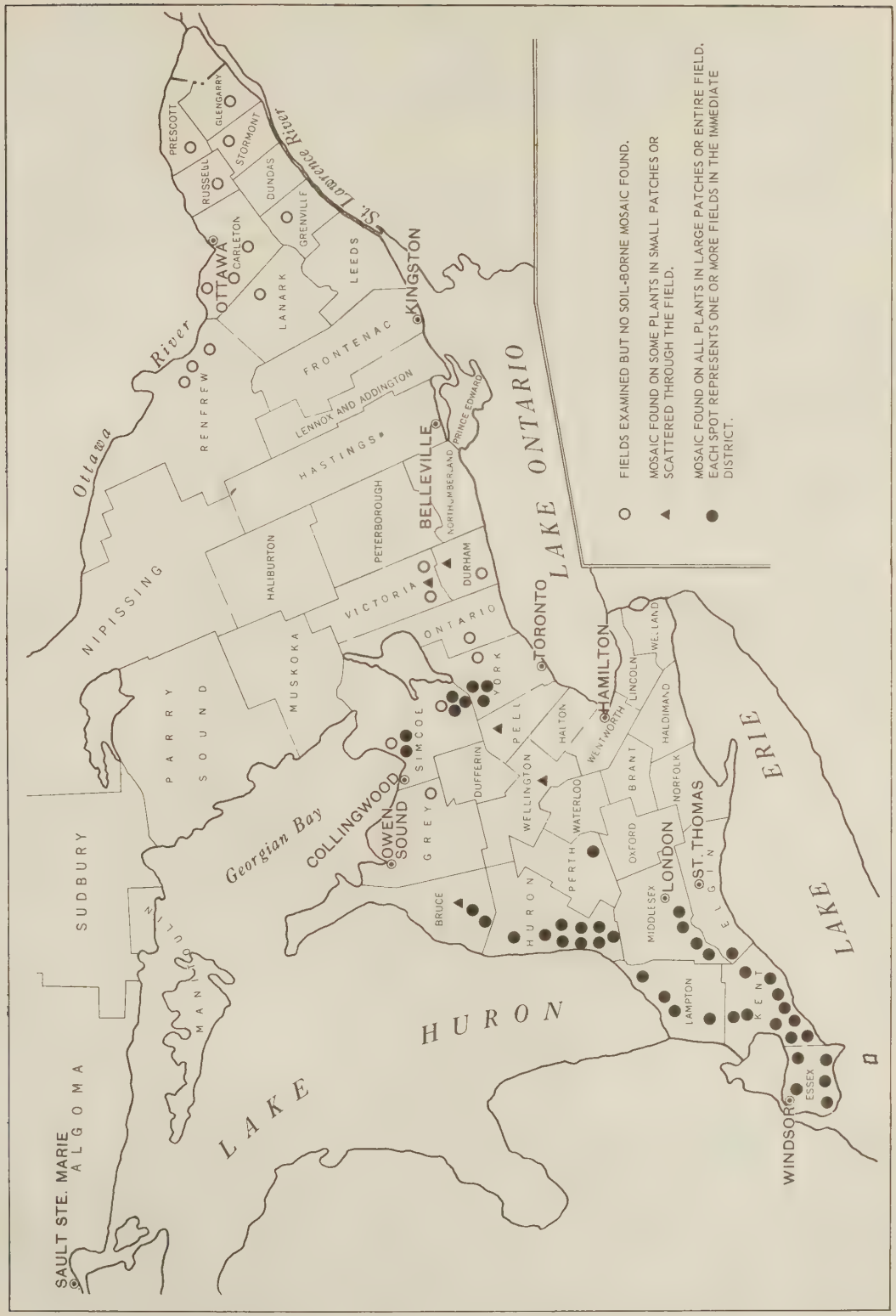
Soils collected from two fields in which mosaic occurred in 1960 were passed through a 1/4 inch screen to remove coarse particles and stones. Lots of 1/3 cu. ft. each were measured, and one lot of each soil was treated with 1 ml of ethylene dibromide, another with 1.5 ml of Telone, and another lot was left untreated. The treated soils were sealed for 1 week in polyethylene sheeting, then aired for one month and placed on top of 3 inches of John Innes potting soil mixture in boxes as described above and winter wheat test varieties were grown in the soil. Mosaic symptoms developed equally on plants in the ethylene dibromide-treated and non-treated soils but no symptoms developed on plants in either of the soils treated with Telone.

(d) Differences between mosaic from Ontario and Illinois soils

Soil from a field in Illinois, U.S.A. where the original type of soil-borne wheat mosaic was first identified (3), was obtained for comparative tests at Ottawa. The reactions of wheat and rye varieties were tested in boxes under the same conditions as used for the tests with Ontario soil. Mosaic symptoms developed on all the wheat varieties tested, but were more severe on Michigan Amber, Pawnee and Kent than on the other varieties. In addition, mosaic symptoms developed on Horton rye grown in the Illinois soil, but not in the Ontario soil. The nature of the symptoms on wheat differed. There were no distinct spots or short streaks but, instead, there was more extensive mottling associated with the Illinois than with the Ontario disease.

Sap transmission tests have been done by rubbing dilutions of sap from diseased plants onto the leaves of Kent, Michigan Amber and Pawnee wheat seedlings, using celite as an abrasive. The plants were incubated at 50°-65°F. Symptoms developed on the plants inoculated with sap from diseased plants grown in the Illinois soil, but not from plants grown in the Ontario soil.

Using the electron microscope, particles that appeared to have a unit length of 140-160 mμ and a width of 25 mμ have been found in sap from diseased plants grown in the Illinois soil, but no particles attributable to a virus have been found in sap from diseased plants from the Ontario soil.


FIELDS EXAMINED BUT NO SOIL-BORNE MOSAIC FOUND.
MOSAIC FOUND ON SOME PLANTS IN SMALL PATCHES OR SCATTERED THROUGH THE FIELD.
MOSAIC FOUND ON ALL PLANTS IN LARGE PATCHES OR ENTIRE FIELD. EACH SPOT REPRESENTS ONE OR MORE FIELDS IN THE IMMEDIATE DISTRICT.
LAKE ONTARIO
LAKE HURON
LAKE ERIE
Ottawa River
St. Lawrence Rapids
Georgian Bay
PRESCOTT
GLENGARRY
RUSSELL
STORMONT
DUNDAS
GRENVILLE
CARLETON
LANARK
LEEDS
FRONTENAC
LENNOX AND ADDINGTON
HASTINGS
PRINCE EDWARD
NORTHUMBERLAND
DURHAM
PETERBOROUGH
HALIBURTON
VICTORIA
ONTARIO
YORK
PEEL
HALTON
WENTWORTH
LINCOLN
WELLAND
HALDIMAND
NORFOLK
BRANT
OXFORD
MIDDLESEX
ELGIN
KENT
ESSEX
LAMBTON
WELLINGTON
DUFFERIN
SIMCOE
GREY
BRUCE
HURON
PERTH
WATERLOO
MUSKOKA
PARRY SOUND
NIPISSING
RENFREW
MANITOULIN
SUDBURY
ALGOMA
OTTAWA
KINGSTON
BELLEVILLE
TORONTO
HAMILTON
LONDON
ST. THOMAS
COLLINGWOOD
OWEN SOUND
SAULT STE. MARIE
WINDSOR

Wheat streak mosaic

Wheat streak mosaic has been found in Canada only in southern Alberta and southwestern Saskatchewan in areas where winter wheat is grown. As indicated previously, the vector, Aceria tulipae K. has been found at Carlyle, Sask., and also at Ottawa, Ontario (10), but wheat streak mosaic has not been found in these areas.

Winter wheat crops were examined in southern Alberta during June 21-23, 1961. Wheat streak mosaic was found only on scattered plants in experimental plots at Lethbridge, and similarly, only a trace to 1 percent of the plants were diseased in fields examined on a route from Lethbridge through Welling, Magrath, Whiskey Gap, Cardston and Hill Spring, and from Lethbridge through Nobleford, and High River to Calgary. The only winter wheat with 100 percent infection was a sparse stand of volunteer plants that had overwintered in a sweet clover crop near High River. The low incidence of wheat streak mosaic in southern Alberta is attributed to the elimination of most immature wheat that could be carrying the virus in early fall, and the delaying of seeding of winter wheat until after the first week of September, both of which are recommended for its control (12).

Wheat spot mosaic

Wheat spot mosaic, a non-sap transmitted virus, transmitted by Aceria tulipae K., is commonly associated with wheat streak mosaic in Alberta (7). The spotting symptoms are usually masked in the field because the infected plants are usually also infected with the wheat streak mosaic virus. The combined infection results in severe streak mosaic symptoms and severe chlorosis. By painstaking mite-transmission tests, the spot mosaic virus has been isolated repeatedly from plants showing such symptoms. In 1961, the presence of spot mosaic was suspected on wheat in the Lethbridge and High River areas of Alberta, but no transmission tests were done to prove it.

A. tulipae has been found on wheat at Ottawa and, although wheat streak mosaic has not been found, a mild disease similar to wheat spot mosaic develops on wheat after mites from field plants have fed on it (10). In 1961, spot mosaic symptoms were found on Kent winter wheat sown in plots in late August and early September, 1960. Unlike the isolates of spot mosaic found in Alberta, the virus isolated at Ottawa caused only mild chlorotic spots and chlorosis, and no severe stunting.

Discussion

Virus diseases that cause mosaic symptoms on wheat in the Prairie Provinces and Ontario are listed in Table 3. Two other viruses that can infect wheat were not included in this report. Barley yellow dwarf virus, which has a wide range of perennial grass hosts, and several aphid vectors, some of which occur in all grassland areas, has been observed by the author or reported by others in all provinces except Newfoundland (4, 15, 16). In 1961 it was observed on wheat in the three Prairie Provinces and Ontario. Barley stripe mosaic virus, which is seed-borne, may be found wherever infected barley seed is grown, but it has not been reported in commercial fields of wheat in Canada.

Table 3. Provinces in which mosaic diseases have been found in wheat.

Mosaic disease	Vector	Alta.	Sask.	Man.	Ont.
Wheat streak	mites	+	+	--	--
Wheat spot	mites	+	--	--	--
Agropyron	?	--	+	--	+
Soil-borne	?	--	--	--	+
Striate	leafhopper	--	+	+	?
"blotchy chlorosis"	?	--	+	+	--

The presence of vectors and reservoir hosts are probably the most important factors determining the distribution of the viruses that cause mosaic symptoms on wheat in Canada. The transmission of wheat streak mosaic virus is dependent on the eriophyid mite Aceria tulipae in southern Alberta and southwestern Saskatchewan. The use of winter wheat and cultural practices that provide a continuous supply of immature wheat, on which the mites can multiply, assures the multiplication of the virus. The same mite becomes abundant on winter wheat that is sown in the spring and remains green through summer and fall, both in southeastern Saskatchewan and at Ottawa, but wheat streak mosaic virus has not been detected at either location. Perhaps it is absent because there are no reservoir hosts in these areas. Conversely, Agropyron mosaic virus, for which no vector has been determined, occurs on Agropyron repens and wheat in southeastern Saskatchewan and Ontario, and in Prince Edward Island. It has not been detected in Alberta even though A. repens, a good reservoir host, is common. Possibly vectors are absent, or for some other reason the virus has not yet spread to that area.

Endria inimica, the leafhopper vector of wheat striate mosaic virus, is common in most grassland areas of southern Canada (1), but the disease has been proven to be present only in southeastern Saskatchewan and southern Manitoba. Perhaps the appearance of the disease in this area was dependent on the extensive use of highly susceptible varieties like Ramsay and Selkirk. It is interesting to note that the disease was first discovered in South Dakota in areas where the highly susceptible varieties of winter wheat, Minter and Nebred were grown. Striate mosaic was not recognized in North Dakota until 1959, but the durum and some hard red spring wheat varieties commonly grown now are susceptible (17). Its occurrence has been suspected in Ontario, but the lack of more conclusive proof of its presence appears to be related to the lack of susceptible varieties that develop good diagnostic symptoms, and the unfortunate use of such varieties as test plants. Striate mosaic was found in Alberta. Tests at Ottawa have shown that Kharkov 22 M.C., the most commonly-grown variety of winter wheat in southern Alberta, is highly resistant. Winalta, a new variety of winter wheat, recently developed for the area, appears to have inherited a high degree of susceptibility from Minter. If this variety is widely used, attention should be directed toward the possible appearance of wheat striate mosaic. However, it is likely that winter wheat sown at the times recommended in southern Alberta would escape infection.

Plants showing the "blotchy chlorosis" symptoms, like plants infected with the wheat striate mosaic virus, were scattered singly among the normal wheat plants. Severely diseased plants were sometimes found closely united with normal healthy plants. Such a situation is not usually apparent with cereal

diseases caused by fungi. Barley yellow dwarf virus may initially infect widely scattered plants but, usually, adjacent plants become infected because of local aphid movement, and the disease develops in patches. On the basis of the distribution of diseased plants as well as the nature of the symptoms, it is suspected that the "blotchy chlorosis" symptoms are caused by a leafhopper-transmitted virus.

Also, on the bases of symptoms and distribution in the field, the mosaic of wheat in southwestern Ontario is suspected to be caused by a soil-borne virus. The association with soil has been proven, but it has not been shown that a virus is the cause. Like the soil-borne wheat mosaic in Illinois, U.S.A., the Ontario disease appears to be favored by the practice growing wheat frequently on the same land, high soil moisture, and long periods of cool soil temperatures. The symptoms do not develop on wheat sown in spring or grown in a warm greenhouse. Not enough is known to suggest why this disease occurs on winter wheat in certain areas of Canada but not in others.

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BARLEY YELLOW DWARF VIRUS SURVEY IN CANADA, 1961[1]

Harvey C. Smith[2]

Abstract

Tests with detached leaves from cereals and grasses showed that Rhopalosiphum padi (L.) was the most efficient vector of barley yellow dwarf virus (BYDV) in samples from Quebec, Manitoba, and Saskatchewan. Both R. padi and Macrosiphum avenae (F.) were efficient vectors of BYDV from Ontario, New Brunswick, and Alberta. R. maidis (F.) was of minor importance as a vector of BYDV in Canada. It was occasionally effective on samples from New Brunswick, Ontario, Manitoba, and Saskatchewan.

No true vector-specific isolates of BYDV were found for R. padi or R. maidis. Vector-specific isolates of BYDV for M. avenae were common only in Ontario, where they occurred mainly in autumn-sown cereal crops. In all cases they were less virulent than isolates transmitted by R. padi. This is probably because of the winter-killing of cereals infected with virulent isolates of BYDV. Aphids were noted in half of the crops surveyed but were abundant in only a quarter of the crops.

The incidence of BYDV was moderate (over 10 per cent) in ten per cent of the crops and, in only one crop, in Quebec, was there a serious loss in yield (over 50 percent). The predominance of yellowing on infected barley and wheat and reddening on infected oats was confirmed in the survey. Leaf purpling was recorded on one Brome grass and two barley samples. The main sources of BYDV infection in the spring, in Ontario, are perennial grasses for R. padi, and wheat, rye and barley for M. avenae.

Introduction

As part of an investigation on the variability of the BYDV disease of cereals, a survey was undertaken with the co-operation of officers of the Canadian Department of Agriculture at different Research Stations and Experimental Farms across Canada. The primary aims of the survey were: (1) to determine the incidence of BYDV; (2) to obtain representative isolates for further study; and, (3) to compare the relative efficiency of the three main vectors, Rhopalosiphum padi (L.), (the bird cherry-oat aphid), Macrosiphum avenae (F.) (=M. granarium), the English grain aphid); and R. maidis (F.) (the corn leaf aphid), in transferring the disease.

Methods

Survey forms, plastic bags, and filter paper for wrapping leaf and stem samples were posted from Ottawa on June 29 to most Experimental Farms and Research Stations in Canada. Ten samples of fresh leaves and stems, from

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cereals and grasses showing stunting, yellowing or reddish-purple leaf discoloration, were requested from each Station. The sample requested was 2 to 3 inches of shoot and base of the uppermost leaf, from one tiller of each plant. The samples were wrapped in moist filter paper and placed in the plastic bags before posting. They all arrived in good condition except for two packages which were delayed more than 10 days before delivery.

Each sample was cut and placed in three plastic dishes containing moist filter paper and healthy individuals of one of the three main vectors; R. padi, (R.P.), M. avenae (M.A.) or R. maidis (R.M.). The aphids were allowed an acquisition feed in the refrigerator at about 15°C for 2 or 3 days. Mature aphids were individually transferred to young (1-to 2-leaf) seedlings of Clintland 60 oats which had been grown in an insect-free greenhouse. Five plants, growing in a 4-inch clay pot, were used for the test with each aphid species. The aphids were placed on the plants which were then immediately covered with either a glass tumbler or a 2-inch diameter cellulose cage. After an infection-feeding period of from 2 to 5 days the covers were removed and the aphids killed by spraying with "TEPP" or nicotine sulphate insecticide. The greenhouses were screened and kept free of aphids by weekly spraying with insecticide. Check plants tested with the stock aphid cultures remained free of BYDV infection.

The severity of infection was first recorded after 17 days and again 20 to 25 days after inoculation. The four grades of infection shown in Tables 1 to 3 were as follows:

0 = No symptoms
\+ = Mild infection on one or two plants
++ = Mild infection on more than two, or severe infection on one or two plants.
+++ = Severe infection on three or more plants.

Results

ONTARIO: The results of tests to transmit BYDV from leaf samples from various sources in Ontario are given in Tables 1 and 2.

Grasses: The most significant feature of the results with grasses was the superiority of R. padi as a vector. It transmitted efficiently from 8 of the 39 grass samples. M. avenae transmitted from only two samples and then less efficiently than R. padi. These results showed that Dactylis glomerata, Phleum pratense, Festuca pratensis, and Poa pratensis were probably the most important pasture grass sources of BYDV in Ontario and confirmed the findings of Slykhuis et al. (10) in 1959 that R. padi was the most efficient vector of BYDV from infected perennial grasses at Ottawa.

In the samples labelled "C.E.F. Roadside" (Table 1), the grasses were growing alongside a crop of oats which showed severe barley yellow dwarf adjacent to the grasses but much less towards the centre of the field. Of the grasses tested tested next to the affected oats, Agropyron repens, Phleum pratense, Echinochloa crusgalli, and Setaria viridis were not infective, but Dactylis glomerata and Poa pratensis were. The only aphid species found regularly on grasses during the summer was R. padi and this aphid was the only vector of BYDV from these grasses. Tests on the oat plants at this location (Table 1) suggested that D. glomerata and P. pratensis were the main sources of virus infection because

Table 1. Barley Yellow Dwarf Virus Survey-1961. Incidence in Ontario

GRASSES			Vectors			OATS (Spring Sown)			Vectors		
SPECIES	Locality	Date	RP**	MA	RM	Variety	Locality	Date	RP	MA	RM
Lolium perenne (2)*	C.E.F. ***	17/4	0	0	-	-	C.E.F. Plots	7/6	+	+++	-
Lolium perenne	C.E.F. Greenhouse	28/4	+++	0	0	Garry	Harrow	4/7	0	++	0
Festuca arundinacea(2)	C.E.F. Plots	17/4	0	0	-	Fundy	Harrow	4/7	0	0	0
Festuca rubra (3)	C.E.F. Plots	17/4	0	0	-	Tioga	Harrow	4/7	0	++	0
Festuca elatior	C.E.F. Plots	17/4	+++	++	-	Bonham	Harrow	4/7	0	0	0
Phleum pratense (2)	C.E.F. Plots	17/4	0	0	-	-	Merrickville	13/7	++	-	0
Phleum pratense	Norwood	12/5	0	0	-	-	Guelph	13/7	++	-	0
Phleum pratense	C.E.F. Greenhouse	28/5	+	0	-	-	Guelph	13/7	++	0	0
Phleum pratense	C.E.F. Greenhouse	28/5	+++	0	-	-	Appleton	13/7	0	++	0
Phleum pratense	C.E.F. Roadside	15/9	0	0	-	-	Lancaster	31/7	0	+++	0
Phleum pratense	Ridgetown	13/7	0	0	0	-	C.E.F. Field	14/9	++	0	0
Poa pratensis (2)	C.E.F. Plots	17/4	0	0	-	-	C.E.F. Field	14/9	++	0	0 0
Poa pratensis	C.E.F. Roadside	15/9	++	0	0	-	C.E.F. Field	14/10	+++	++	+
Agropyron repens	Lindsey	12/4	0	0	0	- (3)	C.E.F. Field	14/10	0	++	++
Agropyron repens	Harrow	12/4	0	0	-	- (2)	C.E.F. Field	14/10	0	0	+
Agropyron repens	C.E.F. Plots	31/7	0	0	-	- (5)	C.E.F. Roadside	14/10	+++	+++	+++
Agropyron repens	C.E.F. Roadside	15/9	0	0	0	- (2)	C.E.F. Roadside	14/10	0	++	-
Dactylis glomerata (2)	C.E.F. Plots	17/4	0	0	-	- (2)	C.E.F. Roadside	14/10	+++	0	-
Dactylis glomerata	C.E.F. Roadside	15/9	++	0	0	-	C.E.F. Plots	14/10	+++	0	++
Bromus inermis (3)	C.E.F. Plots	17/4	0	0	-	-	C.E.F. Plots	14/10	+++	0	0
Bromus sterilis	Ridgetown	13/7	++	+	-	-	C.E.F. Plots	14/10	0	+++	+
Echinochloa crusgalli	C.E.F. Plots	27/6	0	0	0	-	C.E.F. Plots	14/10	0	0	++
Echinochloa crusgalli	Ridgetown	13/7	+++	0	0	-	C.E.F. Plots	14/10	0	++	++
Echinochloa crusgalli	C.E.F. Plots	31/7	0	0	-	- (4)	C.E.F. Plots	14/10	0	0	0
Echinochloa crusgalli	C.E.F. Roadside	15/9	0	0	0						
Setaria viridis	C.E.F. Plots	31/7	0	0	-						
Setaria viridis	C.E.F. Roadside	15/9	0	0	0						
Panicum capillare	C.E.F. Plots	31/7	0	0	-						
Phalaris arundinacea (2)	C.E.F. Plots	17/4	0	0	-						

C * Number of samples tested

** RP=Rhopalosiphum padi, MA=Macrosiphum avenae, RM=R. maidis.

*** C.E.F.=Central Experimental Farm, Ottawa.

Table 2. Barley Yellow Dwarf Virus Survey-1961. Incidence in Ontario

WHEAT (Autumn Sown)			Vectors		
Variety	Locality	Date	RP	MA	RM
-	Lindsey	12/4	0	0	-
-	Barrie	12/4	0	+	-
-	Barrie	12/4	0	0	-
-	Windsor	12/4	++	+	-
-	Harrow	12/4	0	0	-
-	Orono	12/4	0	0	-
-	C.E.F. Plots	25/4	0	++	-
Capelle desprez	C.E.F. Plots	25/4	+	++	-
Hybrid 46	C.E.F. Plots	25/4	0	+++	-
Richmond	C.E.F. Plots	25/6	+	+++	-
Kent	C.E.F. Plots	25/4	+	+++	-
Genesee	C.E.F. Plots	25/4	+	+++	-
CD 1569	C.E.F. Plots	25/4	+	+++	-
-	C.E.F. Plots	25/4	+	+++	-
CD 1569	C.E.F. Plots	7/6	+	++	-
Richmond	C.E.F. Plots	7/6	+++	++	-
Kent	C.E.F. Plots	7/6	0	0	-
-	Guelph	7/6	0	+++	-
Ross	Guelph	7/6	++	++	-
-	Ridgetown	7/6	0	+++	-
Pembina	Merrickville	13/7	+	-	0
-	Guelph	13/7	++	-	0
-	Ridgetown	13/7	++	0	0
Ross	Ridgetown	13/7	0	0	0
Kent	Ailsa Craig	13/7	0	0	0

BARLEY (Autumn Sown)			Vectors		
Variety	Locality	Date	RP	MA	RM
Hudson	C.E.F. Plots	7/6	+	+++	-
Hudson	C.E.F. Plots	7/6	0	+++	-
-	Guelph	7/6	+	+++	-
182	C.E.F. Plots	7/6	0	+++	0
52	C.E.F. Plots	7/6	0	0	-
BARLEY (Spring Sown)					
Vantage	Merrickville	13/7	0	-	0
-	Guelph	13/7	++	0	0
-	Guelph	13/7	0	0	0
Brant	Guelph	13/7	+	0	0
-	Guelph	13/7	++	0	0
-	Ridgetown	13/7	++	+	0
RYE (Autumn Sown)					
	C.E.F. Plots	20/4	0	++	-
	C.E.F. Plots	20/4	0	++	-
	Madoc	12/5	0	++	-
	Holland	12/5	0	0	-
	Hanover	12/5	0	0	-
	Norwood	12/5	0	++	-
	C.E.F. Plots	25/5	+	++	-
	C.E.F. Plots	7/6	0	+++	-

there was a much higher proportion of transmission by R. padi here than in samples from other adjacent oat crops with uniform BYDV infection.

Greenhouse tests on the survival of R. padi and M. avenae on grasses showed that R. padi readily colonized and multiplied on Lolium perenne, Phleum pratense, Agrostis palustris, Alopecurus pratensis, Poa annua, and Poa pratensis, whereas M. avenae survived and multiplied only on Poa annua.

Wheat, Barley and Rye (Autumn-sown crops): When these crops were tested during April and May there was a high rate of transmission of BYDV by M. avenae. Later tests during June and July in the wheat plots at the Central Experimental Farm, Ottawa and in southern Ontario showed an increasing efficiency of transmission by R. padi. These results suggest that autumn-sown cereals provide the main over-wintering source of BYDV which is transmitted by M. avenae in the spring, and confirm the records of Slykhuis et al. (9) who also found M. avenae to be the only vector from autumn-sown wheat and barley in early-season tests. The M. avenae-specific isolates obtained were identical to the EGV-1 isolate described by Rochow (7), and were invariably less virulent than the isolates transmitted by R. padi.

Observations on the incidence of aphid vectors in the field at Ottawa showed that, as in 1959 (10), M. avenae was the first cereal aphid seen. It was first seen in oats and wheat on June 1. R. padi was the first aphid found in wheat and rye, on 18 June; and R. maidis was found about the middle of July. Metapolophium dirhodum, Schizaphis graminium, and Sipha agropyri were not found until late in July on wheat and oats. These observations support the hypothesis that M. avenae is the first vector of BYDV in the spring, obtaining the virus from winter wheat, rye and barley. R. padi probably first infects grasses after leaving its winter host (choke cherry) and brings BYDV infection from the perennial grasses to the cereal crops later in the spring. R. maidis and other vectors multiply later in the season and are able to transmit BYDV brought to the cereal crops by R. padi.

Oats and Barley: (Spring-sown crops) The main feature of the tests with spring oats and barley was the fairly clear cut vector-specificity during June and July in contrast to fall tests. Either M. avenae or R. padi alone transmitted from the samples tested in June and July. In October, however, when R. padi, M. avenae, and R. maidis were all abundant in oats, transmission of BYDV from single plants was commonly obtained by both R. padi and M. avenae, and occasionally by all three vectors. In subsequent greenhouse tests only the vector-specificity of the M. avenae isolates was maintained.

OUEBEC: The results of transmission tests with samples from Quebec are given in Table 3.

Wheat, oats and barley: The main vector of BYDV in 18 of the 21 samples was R. padi. M. avenae gave only poor transmission from two samples which were both efficiently transmitted by R. padi. These results are similar to those obtained with grasses in Ontario. This indicates that the main vector of BYDV in Quebec was R. padi and its source, perennial pasture grasses.

NEW BRUNSWICK: (Table 3)

Oats and Barley: Both R. padi and M. avenae readily transmitted BYDV from the samples received. M. avenae appeared to transmit more efficiently than R. padi from the oat samples but R. maidis transmitted BYDV from only

Table 3. Barley Yellow Dwarf Virus Survey-1961. Incidence in Manitoba, Saskatchewan, Quebec, Alberta, and New Brunswick.

Manitoba and Saskatchewan				Vectors		
Crop	Variety	Locality	Date	RP	MA	RM
Wheat	Durum	Winnipeg	27/6	+++	0	-
Wheat	-	Winnipeg	20/7	+	0	0
Wheat	-	Winnipeg	20/7	++	0	0
Wheat	Thatcher	Swift Current	12/7	++	0	+
Wheat	Pelissier	Swift Current	12/7	++	0	0
Oats	-	Winnipeg	27/7	+	0	0
Oats	Rodney	Swift Current	12/7	0	0	0
Barley	-	Winnipeg	20/7	+	0	0
Barley	-	Brandon	23/7	0	0	+
Barley	-	Brandon	23/7	+	0	+
Barley	Atlas 57	Swift Current	12/7	+++	0	0
Barley	Bonneville	Swift Current	12/7	0	0	+
Quebec						
Wheat	Thatcher	Lennoxville	8/7	+++	+	0
Oats	Victory	Lennoxville	8/7	+++	0	0
Oat	Garry	Lennoxville	8/7	0	0	0
Oats	00.3.2	Lennoxville	8/7	+	0	0
Oats	-	St. Anne (2)	27/7	++	0	0
Oats	-	St. Anne	27/7	0	0	0
Oats	-	St. Anne	27/7	++	+	0
Oats	-	St. Anne	15/9	+++	0	0
Barley	Keystone	Lennoxville	8/7	++	0	0
Oats	Roxton	Lennoxville	8/7	++	0	0
Barley	Montcalm	Lennoxville	8/7	+++	0	0
Barley	Brant	Lennoxville	8/7	++	-	-
Barley	00591	Lennoxville	8/7	+++	0	0
Barley	Len. 30	Lennoxville	8/7	0	0	0
Barley	Le -	St. Anne	27/7	++	0	0
Barley	-	St. Anne (5)	27/7	+	0	0

Alberta				Vectors		
Crop	Variety	Locality	Date	RP	MA	RM
Wheat	Jones Fife	Fort McLeod	22/6	++	++	-
Wheat	-	Welling	22/6	+++	++	-
Wheat	Kharkov	Whiskey Gap	22/6	+++	++	-
Wheat	-	Magrath	22/6	++	++	-
Wheat	-	Lethbridge	27/6	++	++	-
Wheat	-	Lethbridge	27/6	+	0	-
Oats	-	Magrath	22/6	++	++	-
Oats	-	Lacombe	12/7	++	++	++
Barley	-	Magrath	22/6	++	+++	-
Grass	Bromus	Fort Vermillion	27/7	+	0	0
New Brunswick						
Oats	Fundy	Fredericton	11/7	0	+	+
Oats	Rodney	Fredericton	11/7	++	0	+
Oats	CH 5612	Fredericton	11/7	0	0	0
Oats	CH 5612-27	Fredericton	11/7	+++	+++	0
Oats	5246-6	Fredericton	11/7	+++	+++	0
Oats	Fundy	Fredericton	22/7	0	++	0
Oast	Garry	Fredericton	22/7	0	+	0
Oats	Russell	Fredericton	22/7	+++	+++	0
Oats	Shield	Fredericton	22/7	0	+++	0
Oats	Glen	Fredericton	22/7	0	+++	0
Barley	Montcalm	Fredericton	11/7	0	+	0
Barley	Fort	Fredericton	11/7	+++	0	0
Balrye	Herta	Fredericton	11/7	0	0	0
Barley	4675CH1	Fredericton	11/7	++	+	0
Barley	B2M57754	Fredericton	11/7	0	++	0
Barley	Montcalm	Fredericton	22/7	0	+	0
Barley	Fort	Fredericton	22/7	++	0	0
Barley	Carlsberg	Fredericton	22/7	0	0	+
Barley	Chitton	Fredericton	22/7	0	++	++
Barley	Keystone	Fredericton	22/7	0	0	0

two samples of oats and two of barley. In addition to R. padi, M. avenae and R. maidis, Orlob (4) found what S. graminum (Rond.) and Metopolophium dirhodum (Wlk.) were vectors of BYDV in New Brunswick.

MANITOBA and SASKATCHEWAN: (Table 3).

Wheat, oats and barley: (spring-sown) M. avenae did not transmit BYDV from any sample and is therefore probably of little importance as a vector in the field in these provinces. R. padi was an effective vector from 10 of the 13 samples and R. maidis from only 4 of 12 samples tested. As in Quebec, the main over-wintering sources of BYDV are probably the perennial grasses and the main vector, R. padi.

ALBERTA: (Table 3)

Wheat (autumn-sown), oats and barley: R. padi and M. avenae were both important vectors of BYDV from Alberta samples. R. maidis gave good transmission from the one sample tested. The first record of BYDV in Canada was from barley in Alberta in 1955 (2), when a species of Rhopalosiphum was found to be the vector.

Grass: R. padi transmitted BYDV from the one sample of Brome grass tested.

The cereal virus situation in Alberta resembles that in Ontario and New Brunswick where perennial grasses and winter wheat are the main overwintering sources of BYDV for R. padi and M. avenae respectively.

Discussion

Vector-specificity has been widely studied as a possible basis of distinguishing between strains of BYDV. The stability of this characteristic in some isolates was first shown by Toko and Bruehl (12). Rochow (6, 8) subsequently found that the bulk of the BYDV isolates from oats in New York were vector-specific to M. avenae, the English grain aphid, and a very few isolates were readily transmitted by R. padi.

As Bruehl (1) has pointed out, the bulk of the isolates in many other areas of the United States were, however, non-specific and could be readily transmitted by both R. padi and M. avenae. Most vector studies with BYDV have shown that R. padi (also called R. fitchii F. in error (3)) was a more efficient vector of BYDV than M. avenae and produces a more severe disease (9, 12). The only reference to M. avenae-specific isolates (MGV) causing severe infection is that of Smith (11) who found that Saia and Fulghum oats were more severely infected in seedling tests. Field tests, however, showed that these varieties recovered in the adult stage and suggested that the more severe seedling infection was probably due to more efficient transmission of MGV by M. avenae, which is probably better adapted to feeding on Saia and Fulghum than R. padi.

The vector-specificity of MGV isolates was found to be more complete than that of other isolates (7, 13). This could be explained on the basis that M. avenae changes the virus slightly and transmits the less virulent isolates more effectively, but is unable to transmit the less virulent MGV isolates, particularly from oats which are not the best host for R. padi. This hypothesis is supported by transmission experiments using various Canadian isolates of BYDV. In all cases, the

MGV isolates were of reduced virulence and R. padi was rarely able to transmit them from oats or rye, although it did so quite consistently when they were first isolated from wheat. Thus the M. avenae vector-specificity of BYDV appears to have resulted from continuous transmission of the virus between autumn- and spring-sown cereals by one vector, M. avenae. The main reason for the mild or attenuated nature of the MGV isolates in Canada is probably that infection with the more virulent isolates results in winter-killing of infected cereals and hence they can only survive in perennial grasses. The MGV isolates were found mainly in Ontario in autumn-sown wheat, barley and rye, as previously reported by Slykhuis et al. (7).

Isolates transmitted by R. padi were much less specific than the MGV isolates when tested in the greenhouse and were much more virulent when transmitted by either R. padi or M. avenae. They were found predomiantly in Quebec, Manitoba and Saskatchewan on cereals but they were also found in Ontario where they were particularly associated with perennial grasses. Later in the spring, when R. padi became more abundant, they were found more readily on oats and wheat.

Isolates that were transmitted with equal facility by R. padi or M. avenae were found mainly in New Brunswick and Alberta and, in late summer, in Ontario. These isolates had probably resulted from mixed field infections of BYDV when both infective R. padi and M. avenae occurred on the same plant. In some cases, MGV isolates were obtained from these mixed infections, as first suggested by Watson and Mulligan (13).

No true R. maidis-specific isolates were obtained in this survey and all isolates which were originally transmitted efficiently by R. maidis were subsequently transmitted more efficiently by R. padi in greenhouse tests.

Vector-specific strains of BYDV appear to be rare except in Ontario and New York where there is a continuous association of M. avenae as a vector of attenuated strains of BYDV in autumn-sown cereals.

A general conclusion from this survey would be that the most efficient vector or vectors of BYDV in any locality or crop depend primarily on the source of overwintering virus infection and, secondly, on the predominating aphids in each area previous to the samples being taken.

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VEGETABLE DISEASES ON MUCK SOILS IN THE MONTREAL AREA IN 1961

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A survey of the plant diseases which occur on vegetable crops grown on muck soil in the Ste. Clothilde and Sherrington districts was initiated in 1959. It was extended to include other muck soil vegetable-producing areas in 1960. The aim of the annual survey was to determine the most important diseases attacking the crops grown in these areas. It was noted (4, 5) that the same diseases were not important in all the areas surveyed, nor did they necessarily occur in successive years. The scope of the survey does not include an estimation of losses caused by the various diseases; its intention is rather to obtain information on the distribution and intensity of diseases which attack the most important of the vegetable crops grown on muck soils as part of a study of the epidemiology of these diseases. It is hoped that the information obtained can eventually be used to forecast disease occurrence and the necessity of control measures.

For convenience, the muck soils of the Montreal area were divided into 4 regions: Ste. Clothildé, Sherrington, Napierville and Farnham. Observation stations were established in each region, taking into account the principal vegetable crops grown and the acreages involved. The stations were visited from time to time during the summer and records taken of the diseases encountered. The diseases observed in 1961, and their intensity, are presented in Tables 1-4.

Table 1 - Diseases observed in the Ste. Clothilde region

CROP	DISEASES	REMARKS
CARROT (10 Fields)	Alternaria leaf blight (*Alternaria dauci*)	Mod. in 3 fields
	Cercospora leaf blight (*Cercospora carotae*)	Mod. in 3 fields
	Root-knot nematode (*Meloidogyne hapla*)	Sl. to sev. in 3 fields
	Bacterial blight (*Xanthomonas carotae*)	Mod. in 1 field
CABBAGE (1) Field)	Black Leaf Spot (*Alternaria* spp.)	Sl. in 1 field
CELERY (6 fields)	Early blight (*Cercospora apii*)	Tr. to sl. in 3 fields
	Late blight (*Septoria apii-graveolentis*)	Sl. to sev. in 3 fields
LETTUCE (3 Fields)	Downy mildew (*Bremia lactucae*)	Tr. in 1 field
	Drop (*Sclerotinia sclerotiorum*)	Sl. to mod. in 2 fields
ONION (6 Fields)	Downy mildew (*Peronospora destructor*)	Tr. to mod. in 3 fields
	Blast (*Botrytis* spp.)	Tr. to mod. in 3 fields
POTATO (6 Fields)	Late Blight (*Phytophthora infestans*)	Mod. in 4 fields
	Leaf roll (virus)	Sl. in 2 fields

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Table 2. - Diseases observed in the Sherrington region

CROP	DISEASES	REMARKS
CARROT (7 Fields)	Alternaria leaf blight (Alternaria dauci)	Sev. in 3 fields
	Cercospora leaf blight (Cercospora carotae)	Sev. in 3 fields
	Root-knot nematode (Meloidogyne spp.)	Sl. in 1 field
CELERY (7 Fields)	Early blight (Cercospora apii)	Mod. in 3 fields
	Bacterial blight (Pseudomonas apii)	Mod. in 2 fields
	Aster yellows (aster yellows virus)	Tr. in 2 fields
LETTUCE (11 Fields)	Downy mildew (Bremia lactucae)	Sl. in 2 fields
	Drop (Sclerotinia sclerotiorum)	Tr. in 1 field
	Bottom rot (Rhizoctonia solani)	Tr. in 1 field
	Mosaic (virus)	Tr. in 1 field
	Aster yellows (aster yellows virus)	Tr. in 1 field
	Calcium deficiency	Sev. in 3 fields
	Tip burn	Mod. in 2 fields
ONION (9 Fields)	Downy mildew (Peronospora destructor)	Mod. to sev. in 4 fields
	Blast (Botrytis spp.)	Tr. to sev. in 5 fields
POTATO (6 Fields)	Late blight (Phytophthora infestans)	Mod. to sev. in 5 fields
	Blackleg (Erwinia carotovora)	Tr. in 1 field

Table 3 - Diseases observed in the Napierville region

CROP	DISEASES	REMARKS
CARROT (6 Fields)	Alternaria leaf blight (Alternaria dauci)	Tr. in 3 fields
	Cercospora leaf blight (Cercospora carotae)	Sl. in 3 fields
ONION (3 Fields)	Downy mildew (Peronospora destructor)	Tr. in 2 fields
	Blast (Botrytis spp.)	Sev. in 1 field
TURNIP (1 Field)	Downy mildew (Peronospora parasitica)	Sl. in 1 field
POTATO (2 Fields)	Late blight (Phytophthora infestans)	Mod. in 2 fields

Table 4 - Diseases observed in the Farnham region

CROP	DISEASES	REMARKS
CARROT (10 Fields)	Alternaria leaf blight (Alternaria dauci)	Tr. to mod. in 5 fields
	Cercospora leaf blight (Cercospora carotae)	Sl. in 3 fields
	Root-knot nematode (Meloidogyne spp.)	Tr. in 2 fields
LETTUCE (3 Fields)	Aster yellows (aster yellows virus)	Tr. in 2 fields
	Calcium deficiency	Tr. in 1 field
ONION (4 Fields)	Downy mildew (Peronospora destructor)	Tr. to sl. in 2 fields
	Blast (Botrytis spp.)	Sl. to mod. in 2 fields
POTATO (5 Fields)	Late blight (Phytophthora infestans)	Sl. to sev. in 3 fields
	Blackleg (Erwinia atroseptica)	Tr. in 2 fields

Disease index: Trace - 1-10 percent affected plants
Slight - 10-30 percent affected plants
Moderate - 30-60 percent affected plants
Severe - 60-100 percent affected plants

The intensity of leaf blights of carrot (Alternaria dauci and Cercospora carotae) varied from one region to another, being much more severe at Ste. Clothilde and Sherrington than at Napierville and Farnham. Late blight of celery (Septoria apii-graveolentis) was observed only in 3 fields in the Ste. Clothilde region, while onion downy mildew (Peronospora destructor) and late blight of potato (Phytophthora infestans) were observed for the first time since the survey was initiated in 1959. It appeared that weather conditions favorable for late blight were also favorable for downy mildew of onion. The acreage of muck soil infested with the root-knot nematode (Meloidogyne spp.) is increasing. The root-knot nematode in the Ste. Clothilde area has been identified as M. hapla. Blast (Botrytis sp., probably B. cinera) was severe in fields where no fungicide was applied.

Two uncommon bacterial diseases were observed for the first time on muck soils, bacterial blight of carrot (Xanthomonas carotae) and bacterial blight of celery (Pseudomonas apii). The carrot blight occurred in plots at the Ste. Clothilde Experimental Substation in a field where carrots had been grown the previous year. Whether or not the disease was seedborne could not be determined. This disease, according to Conners (2) occurred at La Trappe in 1938. The only other report of its occurrence in Quebec is by Jacuqes (3).

Pseudomonas apii on celery was first reported from Quebec in 1923 (1); it has not been observed again until this year. It was observed at Sherrington in 2 fields transplanted with plants of the variety Utah 10-B grown from the same lot of seed. It could not be found in other fields of the same variety. A greenhouse test is underway to determine whether or not that particular lot of celery seed was infected.

It is hoped that information obtained from this annual survey and from other epidemiological studies underway will be useful in understanding the

different factors that influence disease development on vegetable crops in muck soils. Such information should enable a better timing of fungicidal applications and permit more effective and economical control of vegetable diseases and consequently the expansion of production on muck soil in southern Quebec.

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SOME RECORDS OF PLANT-PARASITIC NEMATODES ENCOUNTERED IN CANADA IN 1961

R.H. Mulvey[1]

The records on which the following report is based were obtained from identifications of nematodes by the Nematology Section, Entomology Research Institute in 1961. The material recorded from interceptions of imported plant material or soil was supplied by officers of the Plant Protection Division, Production and Marketing Branch, Canada Department of Agriculture.

Root-knot Nematodes

The northern root-knot nematode, Meloidogyne hapla Chitwood, 1949, was found on intercepted plant material from several areas in the United States, on rose from Tyler, Texas, from Phoenix, Arizona, and from Monroe, Michigan; on Weigelia sp. from North Collins, New York; on Clematis paniculata from Kansas; on strawberry from Indiana and New York; on tomato from Virginia; on Delphinium sp. (blue bird) from Michigan. It was found in interceptions of rose from Belgium, Denmark, and Holland, and on Berberis thungergii atropurpurea from Holland. In Canada, M. hapla was found on Rosa multiflora from the Downham Nurseries, Strathroy, Ontario, and on Berberis sp. (Sheridan Red) from the Sheridan Nurseries, Toronto, Ontario.

The southern root-knot nematode, Meloidogyne incognita incognita (Kofoid & White, 1919) Chitwood, 1949, was intercepted on importations from the United States; of tomato from Georgia, of Sansevieria sp. from New York, and of rose from Tyler, Texas; on tea roots from Ceylon, and in plant soil which supported ornamentals from Italy. M. incognita incognita was found on Aeschynanthus sp. from Dundas, Ontario, Canada.

The cotton root-knot nematode, Meloidogyne incognita acrita Chitwood, 1949, was found on interceptions of tomato from Georgia, U.S.A. The peanut root-knot nematode, M. arenaria arenaria (Neal, 1890) Chitwood, 1949, was found on interceptions of rose from Texas, U.S.A.; on Berberis thunbergii atropurpurea from Holland, on Hoya cornosa from the British West Indies, and on tomato from a greenhouse at London, Ontario, Canada.

Thames root-knot nematode, Meloidogyne arenaria thamesi Chitwood, 1949, was found on interceptions of rose from Texas, on Clematis paniculata from Kansas, possibly on gardenia from New York, and on Cereus peruvianus from Florida, U.S.A. The Javanese root-knot nematode, M. javanica (Treub, 1885) Chitwood, 1949 was intercepted on importations of tomato from Georgia, and Meloidogyne sp. was found on rose from California, U.S.A.

Cyst-forming Nematodes

The 1960-6 survey for cyst-forming nematodes revealed that the clover cyst nematode, Heterodera trifolii Goffart, 1932, was found in soil which had supported plants of Azalea sp. from Holland, and was prevalent in Canada in

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the Maritime Provinces, Quebec, and Ontario. It was found in soil from the Saint John-Fredericton area in New Brunswick, in Prince Edward Island, and in the Montreal area of Quebec; in the Niagara Falls, Toronto, London and Ottawa areas in Ontario. The clover cyst nematode was found in soil which had supported crops of Ladino clover, oats, and rye in the Vineland Station area in Ontario, and it was found in potato soil from British Columbia.

The sugar-beet nematode, Heterodera schachtii A. Schmidt, 1871, was found on sugar beets from the Lethbridge area in Alberta, Canada. The golden nematode, H. rostochiensis Wollenweber, 1923, was found on interceptions of soil supporting shamrock plants from Ireland, the oat cyst nematode H. avenae Wollenweber, 1924, was found in soil supporting oat and wheat from Woodstock, Ontario, Canada, and the grass cyst nematode, H. punctata Thorne, 1928, was found in imported soil from England, and on Chamaecyparis sp. from Holland.

Tylenchids

Tylenchorhynchus acti Hopper, 1959 was found in Coleus sp. soil from Europe; T. brevidens Allen, 1955 was found on interceptions of Calluna vulgaris from Scotland; T. bursifer Loof, 1959 was found on intercepted shrub and evergreen soil from Holland; T. capitatus Allen, 1955, was found in tobacco soil from the Harrow area, Ontario, Canada; T. clarus Allen 1955, was found on rose from California, U.S.A.; T. claytoni Steiner, 1937 was found in soil supporting tomato plants from Virginia, U.S.A.

Tylenchorhynchus dubius (Buetschli, 1873) Filipjev, 1936 was found on heather from Scotland, in rose shrub soil from Denmark, in Fragaria sp. soil from Latvia, in fruit tree soil from Belgium, in Cineraria sp. soil from England, and on Pelargonium sp., in Crassula sp., and in Cactus sp. soil from Europe. T. macrurrus (Goodey, 1932) Filipjev, 1936 was found in fruit tree soil from Belgium and on interceptions of soil supporting lavender and ornamental shrub plants from various areas in Europe; T. maximus Allen 1955 was found in Canada on oats from the Buckingham area, Quebec, on alfalfa and oats from the Finch area in Ontario, and on white birch from the Vienna area, Ontario. Tylenchorhynchus sp. was found on carnation from Poland.

Tylenchus orbus Andrássy, 1954 occurred in imported lavender and ornamental shrub soil from Europe; Tylenchus sp. (near T. costatus de Man, 1921) was found on importations of giant foxglove from Michigan, U.S.A. and Tylenchus sp. (near T. duplexus (Hagemeyer and Allen, 1952) Andrássy, 1954) was found on carnation imported from Poland.

Stem Nematodes

The potato-rot nematode, Ditylenchus destructor Thorne, 1945 occurred on potato from Ellerslie, Prince Edward Island, Canada. This finding does not represent an extension of its known distribution in the province. Ditylenchus dipsaci (Kuehn, 1857) Filipjev, 1936 was recorded from British Columbia, Canada, on narcissus from Saanichton, on iris and hyacinth from Victoria and on narcissus from Cobble Hill, Victoria. Ditylenchus sp. (now being studied by Dr. L.Y. Wu) was found on hyacinth from Vancouver Island, British Columbia.

Root Lesion Nematodes

Pratylenchus convallariae Seinhorst, 1959 was found on importations of lily-of-the-valley pips from Hamburg, Germany and on Juniperus sp. from Holland; P. minyus Sher & Allen, 1953 was found on carnation from Poland and on white birch from Vienna, Ontario, Canada. Pratylenchus penetrans (Cobb, 1917) Filipjev & Schuurmans Stekhoven, 1914 was found on interceptions of chrysanthemum from Hungary, on rose shrubs from Denmark, in shrub and evergreen soil from Holland, in fruit tree soil from Belgium, in Ribes sp. soil from Holland, on Calluna vulgaris from Scotland. It was found in Canada in potato soil from British Columbia; on Lasalle red clover from the Central Experimental Farm, Ottawa, Ontario, and on strawberry from Hatzic, British Columbia.

Pratylenchus pratensis (de Man, 1880) Filipjev, 1936 was found in soil supporting ornamental plants from Italy, in fruit tree soil from Belgium, in soil supporting Fragaria sp. from Latvia, and on rose from Denmark, and in soil supporting potatoes from British Columbia, Canada.

Pratylenchus, sp. (near P. goodeyi Sher & Allen, 1953) occurred in evergreen soil from Holland; Pratylenchus sp. (near P. irregularis Loof, 1960) was found in imported soil supporting Picea pungens from Holland; Pratylenchus sp. (near P. vulnus Allen and Jensen, 1951) was found in imported evergreen soil from Holland and, Pratylenchus spp. were found on onion from the Okanagan Valley in British Columbia, on spruce seedlings from east of Ottawa, Ontario and in imported Ribes sp. from Holland.

Hoplolaimids

Helicotylenchus erythrinae (Zimmermann, 1904) Golden, 1956 was intercepted on Calluna vulgaris from Scotland, in soil supporting Cineraria sp. from England, and in shrub and evergreen soil from Holland.

Helicotylenchus spp. were found on importations of chrysanthemum from Hungary, on tomato from Virginia, U.S.A., on Hoya carnosa from the British West Indies, in carnation soil from Poland, in ornamental plant soil from Italy, in grass and soil from West Germany, and in Cineraria sp. soil from England. Species were recorded from Ontario, Canada, on oats from Finch, Aeschynanthus sp. from Dundas, spruce seedlings from east of Ottawa, grass sod from Richmond, and on alsike clover from Morewood.

Rotylenchus goodeyi Loof & Oostenbrink 1958 was found in shrub and evergreen soil from Holland, and in cactus oil from Europe. It was recorded from Canada on blue violet from North Gower, Ontario, and on wild strawberry and grass from the La Fleche caverns, Quebec.

Rotylenchus uniformis (Thorne, 1949) Loof & Ostenbrink 1958 was found on interceptions of Picea albertiana canica, Juniperus squamata, and Juniperus sp. from Holland, in shrub and evergreen soil from Holland, in Fragaria sp. soil from Latvia, and on carnation from Poland.

Scutellonema brachyurum (Steiner, 1938) Andrássy, 1958 occurred in imported begonia and gardenia soil from Europe.

Criconematids

Criconemoides lobatum Raski, 1952 was recorded from grass in the west Ottawa and Richmond areas of Ontario, and Criconemoides sp. (near C. informe (Micoletsky, 1921) Taylor, 1936) was intercepted in soil supporting Fragaria sp. from Latvia; Criconemoides sp. (possibly a new species) was found in ornamental soil from Italy.

Paratylenchus spp.

Paratylenchus amblycephalus Reuver, 1959 was found in Ribes sp. soil from Holland. A pin nematode Paratylenchus sp. (near P. microdorus Andrassy, 1959) was found in grass soil from West Germany. A new species of Paratylenchus which is being studied by Dr. L.Y. Wu, Nematology Section, Entomology Research Institute, Ottawa, Canada, was found on intercepted Calluna vulgaris from Scotland.

Paratylenchus sp. (near P. nanus Cobb, 1923) was found in intercepted fruit tree soil from Belgium, in greenhouse plant soil from Europe, in evergreen soil from Holland, and on red clover from the Central Experimental Farm, Ottawa, Ontario, Canada.

Aphelenchids

Aphelenchoides parietinus (Bastian, 1865) Steiner, 1932 was recorded in importations of rape and watercress soil from England. Aphelenchoides sp. (possibly a new species) now being studied by Dr. K.C. Sanwal, Nematology Section, Ottawa) was found on importations of chrysanthemum from Hungary, on lily-of-the-valley pips from Hamburg, Germany, on Picea pungens, and evergreen soil from Holland, on Juniperus sp. from Holland, and on Cumberland raspberry from New Carlisle, Ohio, U.S.A.

Aphelenchus avenae Bastian, 1865 occurred on importations of dahlia tubers from Holland, on Buxus sp. and Ligustrum sp. from Hong Kong, in Ribes sp. soil from Holland, and from pine near La Fleche caverns in Quebec, Canada. It was found in philodendron, Calla sp., and Pelargonium sp. from Europe. Aphelenchus sp. occurred on strawberry from New Carlisle, Ohio, U.S.A.

Seinura sp. was found on importations of carnation, delphinium and giant foxglove from Michigan, U.S.A. and in evergreen soil from Holland.

Dorylamids

Trichodorus christiei Allen, 1957 was found in imported tomato soil from Virginia, U.S.A. T. pachydermus Seinhorst, 1954 was found in evergreen soil imported from Holland. T. primitivus (de Man, 1884) Micoletzky, 1922 occurred in imported Ribes sp. soil from Holland and in fruit tree soil from Belgium.

Xiphinema diversicaudatum (Micoletzky, 1927) Thorne, 1939 was found around the soil supporting roses in a commercial greenhouse at Richmond Hill, Ontario.

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EPIDEMIOLOGY OF POTATO LATE BLIGHT IN THE PROVINCE OF QUEBEC IN 1961

Henri Généreux[1]

The first late blight infection was recorded on July 18 in Labelle Co. Light to moderate infections were noticed in many fields in that district before the end of July. During the last ten days of that month, traces of blight were observed in Napierville, Compton, Drummond, Yamaska and Chicoutimi counties. Traces were also detected on Kennebec at Ste. Francois, Ile Orleans, near Québec City.

Early in August, the disease was observed in many potato fields in Chateauguay, Napierville, Laval, Joliette, Montcalm and L'Assomption counties and unsprayed fields were severely infected by August 12. Traces of blight were recorded on August 1 at Lennoxville and, twelve days later, a 50 per cent infection was recorded. It was also observed on August 12 in South Gaspé and, a week later, the disease was found in a few scattered fields in the Baie des Chaleurs region. It was also recorded on August 14 near Baie Comeau.

From Québec to Gaspé, only traces of blight were detected at the end of August, particularly in a few scattered fields in Levis, Bellechasse, Kamouraska and Bonaventure counties. Elsewhere in the province, the disease was well disseminated and the degree of infection varied from light to moderate. Weather conditions in August favoured the spread of blight. Precipitation totals were twice the normal in the Eastern Townships and the upper part of the St. Lawrence River Valley, whereas in the Gaspé Peninsula and in the Lake St. John district, precipitation excesses ranged from forty to eighty per cent.

By September 8, potato late blight was severe and general in most regions of Québec. Unsprayed fields were already killed in the Eastern Townships, Montreal, Lake St. John and Chicoutimi districts. Tuber rot had been observed in Laval Co. and a few fields had up to 30 per cent of infection by September 10. The foliage of most resistant seedlings and varieties was killed by the disease in the above-mentioned regions. At that time, the spread of blight had also made some progress in the Lower St. Lawrence district.

According to potato inspectors, the potato crop was greatly reduced in the Labelle and Gatineau districts following the early appearance of blight, but only traces of tuber rot were recorded. Generally, the disease did not reduce the potato crop in the Eastern Townships and the Montreal districts. However, heavy losses were recorded in the Lake St. John and Chicoutimi districts. Tuber rot was also found in most of the fields grown in the Lower St. Lawrence area and was especially severe on heavy soils. The Keswick variety, cultivated on a large scale in the Matapedia Valley, was badly infected after digging time.

The following table indicates the extent of blight infection observed on resistant seedlings tested in the regional trials and sprayed with fungicides.

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Table 1. Incidence of late blight of potato seedlings in regional trials.

	Ste. Clothilde		L'Assomption		Peribonka		Ste. Foye
Seedlings	% blight	% tuber rot	% blight	% tuber rot	% blight	% tuber rot	% tuber rot
F5025	15	nil	100	nil	tr.	nil	tr.
F5609	7	"	92	"	nil	tr.	nil
F5649	30	"	100	"	31	nil	tr.
F52100	12	"	100	"	5	tr.	tr.
F5350	8	"	100	"	3	nil	tr.
F4724	16	"	100	"	5	"	nil
F5552	tr.	"	42	"	nil	nil	nil
F4519	10	"	100	"	nil	tr.	tr.

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DISEASES OF FIELD BEANS IN WESTERN ONTARIO IN 1961

G.H. Clark and R.N. Wensley[1]

Surveys were conducted in western Ontario during 1961 to determine the incidence of bean diseases and estimate the damage they caused. The diseases encountered, and their relative importance, are listed below.

Sunscald (nonparasitic). When most bean varieties are approaching maturity, sunscald may occur on any parts of the plant exposed to the sun. On some bush-bean varieties this disorder may appear as early as two to three weeks before maturity. In 1961 sunscald was most severe on the early-maturing varieties, Sanilac and Seaway, planted in late May or early June, and least severe on the late-maturing variety, Michelite. The prevalence of sunscald and the consequent defoliation and abortion of the upper pods was responsible for most of the concern expressed by bean growers this year. On the basis of field observation and reports from bean growers, an estimated loss of ten bushels per acre was incurred in fields severely affected by this disorder.

White Mold (Sclerotinia sclerotiorum) caused more damage than usual in Ontario in 1961; humid conditions in July and August being conducive to its development in some areas. It was most severe where vine growth was heaviest. An estimated loss of five to ten bushels per acre occurred in severely diseased fields. The bacterial blights, (Xanthomonas phaseoli and Pseudomonas phaseolicola) were widespread on all varieties in Ontario and most apparent on the early-maturing variety, Sanilac. However, in the aggregate, they did not cause appreciable damage.

Root Rot (Fusarium solani f. phaseoli) was present in many fields but was generally of only slight economic importance. It occurred chiefly on beans grown in heavy clay soils or in low areas which were flooded during the growing season. An estimated loss of fifteen to twenty bushels per acre occurred in a few severely affected fields.

Rust (Uromyces phaseoli) was found only rarely and infections were not severe.

Mosaic (virus) was of rare occurrence and was seen mainly on Michelite and several unlicensed varieties. The low incidence of mosaic in 1961 may be attributed to the paucity of vectors.

The results of these surveys indicate that sunscald and Sclerotina wilt and rot caused the most reduction in yield of white beans. Although root rots and bacterial blights continue to threaten, they were not the most important diseases of beans in western Ontario in 1961.

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A SURVEY FOR BACTERIAL BLIGHT IN REGISTERED FIELD BEAN CROPS IN SOUTHWESTERN ONTARIO

M.D. Sutton, W.L. Seaman, and V.R. Wallen[1]

Thirty-five bean fields, representing 540 acres grown for registration, were inspected twice during the summer of 1961. The first inspection was made early in July to estimate the prevalence of seed-borne infection in the crop and to distinguish, if possible, the symptoms of halo blight from common blight in the field. The second inspection was made in late August to determine the extent of infection in the crop before harvest. The percentage of pod infection was used during the second inspection as a criterion for disease diagnosis. Twenty-one fields of Sanilac, 9 fields of Michelite and 5 fields of Seaway were represented in the survey. Infected leaves and pods were excised during the second inspection for bacteriological examination in the laboratory.

Sixteen of the fields selected for inspection were sown with the growers' own registered seed from the 1960 crop; eight were sown with seed imported from the United States, and eleven fields with seed purchased from seed merchants in the district. Five lots in the last category were breeders' stock produced at the Ridgetown Agricultural School.

The results of the survey indicate that there has been a build-up of seed-borne inoculum of both Xanthomonas phaseoli (common blight) and Pseudomonas phaseolicola (halo blight) in southwestern Ontario. Of the fields examined, 6 were healthy; 8 had leaf and stem infection but no pod infection, and the remaining fields all contained infected pods. Of the 8 fields sown with imported seed, 4 were free of bacterial disease and the remaining fields showed only a trace of infection. Of the 5 fields sown with breeders' stock, only 2 had slight infection. All 16 fields sown with growers' own seed contained pod infections ranging as high as 25 per cent.

Bacteriological examination of plant material from 15 fields, chosen at random, produced 4 isolates of Ps. phaseolicola and 7 isolates of X. phaseoli. The pathogens were detected and identified by the rapid phage plaque count method. The application of this method confirmed the field diagnosis that both pathogens were present in the crop.

Other bean diseases were noted in the course of the survey. A trace of fuscus blight, Xanthomonas phaseoli var. fuscans was seen in a field of Sanilac and common and yellow mosaic were prevalent in many of the fields. Anthracnose, Colletotrichum lindemuthianum, was not observed.

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1961 PEA DISEASE SURVEY IN THE OTTAWA VALLEY

V.R. Wallen

Eleven fields, comprising 90 acres of field peas, were inspected for the incidence of disease in the area between Ottawa and Renfrew, Ontario.

Although conditions were very favorable for the development of leaf and pod spot (Ascochyta pisi), the disease was detected only as a trace infection in one small field. Its absence in 1961 can be attributed directly to the lack of seed-borne inoculum. The seed produced in 1960 was free of A. pisi because of the extremely dry conditions that prevailed during the summer of that year. The absence of leaf and pod spot in a year favorable for its development is strongly indicative that the seed-borne phase is very important in the epidemiology of this disease.

The finding of anthracnose (Colletotrichum pisi) was of interest. It occurred in trace amounts in several fields. Anthracnose has been rarely reported in Canada and has not been observed for a number of years.

Mycosphaerella blight (Mycosphaerella pinodes) and foot rot caused by Ascochyta pinodella were each noted in trace to moderate amounts in several fields. The Ascochyta foot rot caused 25 percent damage in one field. Rust (Uromyces fabae) occurred sporadically in several fields. Both leaves and stems showed trace to moderate infection. Blight and Rot caused by Sclerotinia sclerotiorum was recorded in two fields. Damage was slight at the time of inspection, but by harvest time, under optimal conditions, severe losses could occur.

Virus diseases were observed, particularly in fields on the Central Experimental Farm, Ottawa. One small field of the variety Arthur was severely affected by streak; 50 per cent of the plants in this field failed to set seed. Another small field of Creamette was infected with both streak and mosaic in trace amounts.

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STORAGE DISEASES OF TOMATOES IN NOVA SCOTIA IN 1961

C. L. Lockhart

A survey was made of the organisms causing rots of tomatoes, varieties Harrow and Stokesdale, in common storage in Nova Scotia. For this purpose, counts were made of the rots as the tomatoes were removed from storage from September to November. A total of 4600 tomatoes were examined. The results are given in Table 1.

Table 1. Rots of stored green-mature tomatoes expressed as percent

Organism	Stokesdale	Harrow
	%	%
Colletotrichum coccodes	12.5	28.5
Alternaria tenuis	2.4	1.6
Botrytis cinerea	0.1	0.3
Phoma destructiva	0.1	0.0
Sclerotinia sclerotiorum	0.2	0.0
Fusarium spp.	0.1	0.1
Penicillium spp.	0.3	0.0
Mucor sp.	0.1	0.0
Bacteria	1.0	1.5
Total rots	16.4	32.0

The variety Harrow appears to be much more susceptible to anthracnose than is the older variety Stokesdale.

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1961 SEED TREATMENT SURVEY IN THE PRAIRIE PROVINCES

F.J. Greaney[1]

The questionnaire type survey, begun in 1959, to determine the extent of the use of treated seed of wheat, oats, barley and flax in the Prairie Provinces, was continued in 1961. The results of the survey, presented in Tables 1 and 2 show that it is evident that there has been little change in the seed-treatment picture in any of the three Prairie Provinces in the past three years. They emphasize, however, the need for more intensive seed-treatment campaigns to encourage many more farmers in Manitoba and Saskatchewan to practice seed treatment.

Table 1. Percentage of total acreages planted with treated seed, 1959-1961.

Province	Wheat			Oats			Barley			Flax		
	1959	1960	1961	1959	1960	1961	1959	1960	1961	1959	1960	1961
Man.	47	48	46	26	25	23	42	42	37	40	41	42
Sask.	60	62	60	61	60	59	75	76	74	63	56	57
Alta.	86	86	85	81	81	80	91	88	88	81	76	74
Pr. Prov's.	68	69	67	65	64	63	78	77	75	66	60	60

Table 2. Results of 1961 seed treatment survey.

Province and Crop District	Percentage of total acreage planted with treated seed:			
	Wheat	Oats	Barley	Flax
MANITOBA				
Average 1/	46.4 (109)	22.6 (106)	36.6 (108)	41.5 (105)
SASKATCHEWAN				
1A	50.8	25.8	50.0	59.3
1B	56.9*	46.9*	72.5*	67.9*
2A	66.8	49.0	71.8	45.3
2B	62.4	49.6	79.9	56.3
3AN	62.9	58.2	76.7	62.8
3AS	72.2	65.4	79.2	68.9
3BN	72.7	69.7	83.2	54.2
3BS	61.8	74.0	75.7	65.0
4A	76.3	70.9	78.3	58.8*
4B	79.4	79.5	83.6	67.5
5A	25.0	27.1	40.4	16.3
5B	29.5	33.0	54.0	41.5
6A	59.0	67.1	78.3	58.7
6B	75.8	72.5	86.4	53.6

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Results of 1961 seed treatment survey (cont'd).

Province and Crop District	Percentage of total acreage planted with treated seed: Wheat	Oats	Barley	Flax
SASKATCHEWAN				
7A	81.0	83.1	91.6	70.1
7B	77.1	78.7	88.7	81.8
8A	38.4	33.4	52.5	35.9
8B	46.0	73.8	76.7	58.3
9A	60.3	66.4	78.9	50.6*
9B	40.2	49.4	65.8	53.3*
Average 1/	59.9 (671)	59.3 (658)	73.7 (667)	57.3 (526)
ALBERTA				
1	84.7	84.5	83.8	69.1
2	91.2	85.5	92.6	79.9
3	88.9	80.2	89.7	78.8
4	79.5	77.5	84.3	56.9
5	74.1	71.4	79.1	75.0*
6	81.4	78.8	87.5	62.5*
7	89.2	78.3	88.2	66.7
Average 1/	85.0 (382)	80.3 (381)	87.6 (380)	74.1 (233)
PRAIRIE PROVINCES				
Average 1/	66.9 (1162)	62.9 (1145)	74.8 (1155)	59.9 (864)

*Based on less than 10 individual reports. 1/ Weighted
The figures in brackets represent the number of individual reports used to determine the percentage acreage figures.

CEREAL DISEASES ENCOUNTERED AT ILLUSTRATION STATIONS IN NORTH AND CENTRAL ALBERTA IN 1961

W.P. Campbell, L.J. Piening and D.W. Creelman

In August, 1961, cereal plots at nineteen locations in central and northern Alberta were examined for the presence of disease. The results of this survey are summarized below.

Barley Diseases

Net blotch (Drechslera teres) and scald (Rhynchosporium secalis) were the most severe and widespread of the diseases observed on barley. Net blotch was found at all the sixteen stations where barley was grown and the intensity of infection ranged from trace on three varieties at Buffalo Head Prairie to moderate-severe on nine of ten varieties at Vermilion. Scald occurred at fifteen of the sixteen stations with the lowest infection ratings being recorded at High Prairie and the highest ratings at Cheddarville and Vermilion where eight of the nine varieties grown were moderately to severely attacked.

Common root rot (Bipolaris sorokiniana and Fusarium spp.) was recorded in trace amounts at most stations. Moderate infections were recorded at Olds, Vermilion, Vegreville and Athabasca.

Loose smut (Ustilago nuda) was seen as trace infections at eight of the sixteen stations. The only variety seriously affected was H53-1409 which had a ten per cent infection at Cheddarville and was five per cent infected at Leslieville and Vermilion. Bacterial blight (Xanthomonas translucens) was recorded at five stations in central Alberta with the heaviest infection occurring at Vegreville. Speckled leaf blotch (Septoria passerinii) was moderate to severe on six of ten varieties at Vegreville and trace on one of eight varieties at Keg River and High Prairie. Ergot (Claviceps purpurea) was recorded only at Vegreville and there only as a trace infection.

Generally, barley diseases were more serious on the stations in central than on those in northern Alberta.

Oat Diseases

The non-parasitic diseases, blast and gray speck (manganese deficiency), were the most common oat diseases encountered. Blast was recorded at seventeen of the nineteen stations, mostly in trace to slight amounts though moderate to severe symptoms were seen at High Prairie. Gray speck occurred at nine locations and it, too, was moderate to severe at High Prairie.

Halo blight (Pseudomonas coronafaciens) was seen in trace to slight amounts at fourteen stations. Leaf blotch (Drechslera avenacea) was trace to slight at nine stations. Trace infections of common root rot (Bipolaris sorokiniana and Fusarium spp.) were recorded at four stations and loose smut (Ustilago avenae) was seen in one variety at Cheddarville.

Wheat Diseases

Common root rot (Bipolaris sorokiniana and Fusarium spp.) occurred in trace to slight amounts at all the nineteen stations visited. It was by far the most common disease observed on wheat, although damage appeared to be very slight.

Glume blotch (Septoria nodorum) was recorded at five stations, mostly as trace infections. It was not observed in the Peace River district. Speckled leaf blotch (Septoria tritici) was seen as trace to slight infections at Wanham and Cheddarville. Trace infections of ergot (Claviceps purpurea) occurred at Evansburg and Vegreville; leaf rust (Puccinia recondita) was trace on two varieties at Vegreville, and loose smut (Ustilago tritici) was slight on one variety at Buffalo Head Prairie.

Flax Diseases

Diseases of flax were not severe on the illustration stations visited. Wilt (Fusarium oxysporum f. lini) was seen in trace amounts in four of eight varieties at Manning and in three of ten varieties at Keg River. Slight to moderate infections of rust (Melampsora lini) were recorded on Redwing at Blueberry Mountain and trace amounts were observed on the same variety at High Prairie. Rhizoctonia stem canker was trace on seven of the twenty-seven varieties in the Co-operative tests at Beaverlodge and was seen on one variety at Blueberry Mountain. There were no flax diseases at six other stations in the Peace River district.

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ALFALFA DISEASES IN CENTRAL BRITISH COLUMBIA IN 1961

E.J. Hawn

The alfalfa disease picture in south-central British Columbia has changed considerably in certain aspects during the last five years.

The expanding use of alfalfa varieties resistant to bacterial wilt (*Corynebacterium insidiosum*) has relegated that disease from one of major to one of minor importance in British Columbia's irrigated stands. It was found in 34 per cent of the fields examined in the Creston, Glenwood, Midway, Boundary, Osoyoos, Kelowna, Vernon, Armstrong, and Kamloops districts in 1961. This compares favorably with an incidence of 78 per cent recorded for 1956.

Crown bud rot (*Rhizoctonia solani*, *Fusarium roseum*, *Ascochyta imperfecta*) was the most prevalent disease in 1961, particularly on irrigated land in the above-mentioned areas. Alfalfa, especially in the ranching areas north of Kamloops, was severely thinned out after four years of growth. A comparison of such stands with those on the Tranquille Farm at Kamloops (where no grazing is permitted) strongly indicated that grazing and tramping contributed materially to cold injury and hence to an accelerated development of crown bud and crown rot during the growing season.

Other diseases noted included downy mildew (*Peronospora aestivalis*), common leaf spot (*Pseudopeziza trifolii* f. sp. *medicaginis-sativae*), yellow leaf blotch (*Leptotrochila medicaginis*), black stem (*Ascochyta imperfecta*) and witches' broom (virus). The importance of these diseases was assessed as slight, placing them in the same general category as that recorded in 1956.

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RAPE DISEASES IN SASKATCHEWAN IN 1961

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The occurrence of rape diseases in 1961 reflected the extremely dry weather conditions: fungus diseases were of negligible importance in the brown and dark-brown soil zones on the prairie and only slight, or occasionally moderate, infections occurred in the black soils of the parkbelt in northern Saskatchewan. The estimated rape acreage in the province in 1961 was 448,000 with an average yield of 550 pounds per acre. This compares with 727 pounds in 1960 and 848 pounds in 1959. The reduced yield is attributed to above-normal temperatures and well below-average rainfall.

White rust (Albugo cruciferarum) was conspicuous only in the Meadow Lake region on the northern fringe of the rape-growing area. Both the conidial and the oospore (hypertrophied) stages were less severe than in 1960 although distribution was general. The general occurrence suggests that the disease is well established in that area and that there was a good carry-over of inoculum. Experimental plots at Saskatoon were inoculated, when seedlings were emerging, with broken pieces of material bearing oospores. Those plots which were watered intermittently by overhead sprinkling during the summer developed slight infections bearing both stages of white rust. None were observed in inoculated, but unwatered plots nor in farmers' fields in the vicinity of Saskatoon. No downy mildew (Peronospora parasitica), either alone or in combination with white rust, was observed in 1961.

During late August surveys, black blight or ring spot (Mycosphaerella brassicicola) was found to be developing well on rape stems in the Meadow Lake area and, to a lesser extent, in the northeast portion of the province. There was copious oozing of spermatia but no perithecia were found. The disease was absent in the dry prairie. Affected stubble from two northern areas was collected in May. No oozing of spermatia from the spermagonia was observed, suggesting that the spermatia are virtually all discharged in the fall or very early spring.

Only one trace infection of stem blight (Sclerotinia sclerotiorum) was seen. Low soil moisture was probably the factor that controlled this disease. Leaf blight, caused by species of Alternaria was present in trace amounts in the northern areas of the province. In experimental plots at Melfort, rape was more affected by Alternaria spotting than was commercial mustard.

Trace to slight infections of aster yellows were found in many northern fields. Traces were also found further south where fields matured earlier. The disease incidence showed a slight increase over that of the last four years. Powdery mildew (Erysiphe polygoni) was severe in two experimental greenhouses at Saskatoon. A heavy infection also developed late in the season in overhead-irrigated plots. This disease could be damaging to crops in seasons favorable to its development.

Black leg (Phoma lingam) was difficult to detect in field collections although occasional pure cultures of the organism could be obtained from suspected material. Discolored rape stubble from the northern areas of the

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province were scattered over experimental plots at seeding time and supplementary overhead watering was applied to the plots in the absence of rain. There was a copius development of pycnidia on the basal stem portions of the rape plants by the end of the season and pure cultures of P. lingam were readily obtained. This confirms our earlier contention that black leg is present on rape in the northern areas and may become serious in wet seasons.

One rape field at Brancepeth developed pitted, spongy, subspherical swellings up to three-quarters of an inch in diameter at the bases of the stems. The field was adjacent to a wheat field which had been sprayed with 2,4-D earlier in the season. Damage was moderate near the sprayed wheat field but became progressively less as the distance from the field increased. The galls were examined for the presence of Plasmodiophora brassicae and nematodes, but neither were found.

Rape seed from the 1961 crop in Saskatchewan should be virtually free of seed-borne diseases if grown on the prairie, and that from the parkbelt should be carrying less disease than ever before.

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TOBACCO DISEASES IN ONTARIO IN 1961

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The most common seedbed disorder in 1961 was damping-off or bed rot caused by species of Rhizoctonia and Pythium. The disease is characterized by a wet, slimy rot of seedlings at the ground level. Fortunately, however, it occurred only in small patches in greenhouses in areas where plants were crowded or the soil remained wet due to poor drainage. The overall losses were estimated to be less than five per cent.

The second most common disorder in seedbeds was yellow patch, caused by an accumulation of inorganic salts and the addition of too much fertilizer. It was most noticeable in the early part of the season, just after the seed had germinated. The yellow, stunted seedlings, which characterize the disorder, often recover after the excess salts are leached out.

A few cases of black root rot (Thielaviopsis basicola) occurred in seedbeds. Its occurrence was found to be mainly due to improper steaming of the soil and to the use of soil fumigants rather than steam. Again, the overall losses were small.

Blue mold or downy mildew (Peronospora tabacina) has, in the past, inflicted heavy damage to tobacco in seedbeds. However, in the past seven seasons losses from this disease have been negligible, mainly because of the preventative spray programs practiced by growers in the United States and Canada. Because blue mold can attain epidemic proportions almost overnight it remains a constant and serious threat to seedling production and is still considered to be the most destructive of all tobacco diseases. Although the disease was not observed in Canada in 1961, a regular spray program is still recommended and followed by most growers.

The most outstanding feature of the tobacco disease picture in the field in the early part of the 1961 season was the occurrence of a disorder known as sore shin. This disorder is characterized by brown to black lesions on the stem, usually on one side and at the ground line. Affected plants remain stunted and usually topple over in a strong wind. The organisms most commonly isolated from the lesions are species of Pythium and, sometimes, Rhizoctonia. Sore shin is most serious during cold, wet springs and in 1961 at least ten per cent of the tobacco fields had to be replanted.

Because of the cold, wet spring, black root rot (Thielaviopsis basicola) caused considerable stunting of plants early in the season. It was more common than in 1960 and even tobacco varieties which have been considered to be highly tolerant were affected. Some recovery occurred later in the season with the advent of warm weather and actual losses were small. The new flue-cured variety "Yellow Gold" was observed to be highly resistant and exhibited very few root lesions. Brown root rot, associated with injury by Pratylenchus species, caused a moderate amount of damage in 1961. The use of soil fumigants, which are being increasingly used, will control this disease.

Weather fleck, characterized by brown, necrotic spots on the leaves, continues to be one of the most serious disorders of flue-cured tobacco in the

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field. Although air pollution is suspected to be the most important causal factor, the precise causes of the disorder are still unknown. The development of tolerant varieties appears to be the only economical means of controlling weather fleck and new varieties are tested as soon as they are developed. In 1961, the variety Delhi 61 was planted on a limited scale on farms that are usually severely affected. It was found to be moderately tolerant and showed considerably less flecking than the more susceptible varieties.

Brown leaf spot, caused by Alternaria species, is also rapidly becoming a serious field disease in Ontario. In the past it was usually confined to maturing leaves, but more recently it has been found on plants at all stages of growth. As some doubt exists as to the actual species of Alternaria that cause the disease in Canada, a study is in progress at Harrow that will attempt to clarify this question. Over ninety isolates of Alternaria have been obtained from diseased tobacco leaves and these are being tested for their ability to produce necrotic spots on tobacco. No control measures have been recommended to date.

Tobacco is susceptible to a very large number of virus diseases. Fortunately, under the conditions that prevail in Ontario, only a few scattered plants are affected and losses from virus diseases are negligible. Viruses observed in 1961 were: Tobacco mosaic, etch (on burley only), cucumber mosaic, streak, ring-spot, alfalfa mosaic, curly-top, potato Y and mottle.

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PLANT-PARASITIC NEMATODES IN SOUTHWESTERN ONTARIO IN 1961

W.B. Mountain and R.M. Sayre[1]

A total of 290 soil and root samples were submitted for diagnosis to the Nematology Section, Research Station, Harrow, in 1961 by provincial extension officers and growers. This is the largest number on record. In general, the root lesion nematode, Pratylenchus penetrans and the northern root-knot nematode, Meloidogyne hapla, were the predominant forms encountered.

The root lesion nematode, Pratylenchus penetrans was again widely distributed on flue-cured tobacco in the Delhi area. A total of 126 samples were submitted for examination. The importance of this nematode in the production of flue-cured tobacco can be gauged by the increased use of nematicides for its control; from approximately 50 acres treated in 1958 to 1500 acres in 1961. Sixty-five samples of eggplant received from the Harrow-Leamington district were all infested with P. penetrans. Samples from five greenhouses of chrysanthemums and three fields of tomatoes in the Leamington area showed high populations of the same nematode.

The incidence of the northern root-knot nematode, Meloidogyne hapla, in southwestern Ontario continues to increase. A greater number of the samples submitted in 1961 were found to be infested with this nematode and new areas of infestation were found in widely separated areas in the province.

For the first time in many years M. hapla was found in fields of flue-cured tobacco near Delhi. This nematode was known to occur in the area prior to the introduction of a rye-tobacco rotation (3) but it has not been reported on tobacco since 1943 (1). In 1961, six tobacco samples from Mt. Brydges and two from Houghton were moderately to heavily infested with M. hapla. The nematode is not expected to occur in a rye-tobacco rotation since rye is an immune crop, but it is likely that vetch and other weeds that occur in many rye fields would allow a build-up of nematode populations. The economic importance of the root-knot nematode in flue-cured tobacco could not be assessed because of the invariable presence of Pratylenchus penetrans.

The degree of infestation of M. hapla on carrot in the Thedford, Jeanette's Creek and Bradford Marshes was much the same as in 1960(2). However, additional areas of infestation were found at all locations. The severity of damage to carrots appears to depend on the preceeding crop, being most severe following potatoes and least severe following onions. Five acres of carrots on sandy soil near Alliston were severely damaged by M. hapla.

Seventeen samples of eggplant from the Harrow-Leamington area were slightly to heavily infested with M. hapla. Although onions are not usually affected by this nematode, two acres of onions, following a crop of potatoes, were severely stunted by a heavy infestation.

The southern root-knot nematode, Meloidogyne incognita, is the predominant nematode species in greenhouses in southwestern Ontario. This pest occurs each year on cucumbers, tomatoes and lettuce. In 1961, tobacco seedlings in one greenhouse at Leamington were infested with M. incognita.

Since the discovery of the bulb and stem nematode, Ditylenchus dipsaci, in the Leamington Marsh in 1957, annual surveys have been carried out to determine its status. In 1961, a survey of fifteen farms that had previously

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been found to be infested showed that a total of nine acres on three of the farms remain infested. In each case, the grower had ignored the control recommendations and had continued to plant onions each year. Those growers who had followed the recommended two-year rotation with a non-susceptible crop harvested onions in 1961 that were completely free of nematode injury. This survey has confirmed the findings of laboratory and greenhouse studies at Harrow that D. dipsaci cannot survive in soils where non-host crops are grown for two seasons.

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